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Brain Size, Grey Matter and Race —Fact or Fiction?

A Reply to Phillip V. Tobias

By SANDY GELLIS

ABSTRACT

A critical review is given of those factors alleged by Dr Phillip V. Tobias to be accompanied by variation in brain weight—such as, sex, size, age of death, nutritional state in early life, source of the sample, cause of death, anatomical level of severance, presence or absence of cerebrospinal fluid, meninges, and blood vessels. Valid comparisons should take sex, occupational group or social class, presence of cerebrospinal fluid, or meninges into account. Most published studies have taken all relevant variables, as well as all or nearly all the unnecessary factors advanced by Dr Tobias into consideration, despite Dr Tobias's claim to the contrary. Most published interracial comparisons are valid. Published evidence exists on the histological, chemical, and functional counterparts of big and small brains. Published interracial comparisons of thickness of the cerebral cortex, including the supragranular layer, are valid, technically and otherwise. There is acceptable data that the cortex of Negroes is thinner in parts and as a whole than that of North Europeans. It is concluded that statements have been based on substantial evidence.

INTRODUCTION

Since the latter part of the eighteenth century, investigations into the neurology of human races have turned in regular and abundant information on the cerebral characters of the major territorial groupings of humanity (Tobias, pp. 3-5). By the early part of this century, an enormous amount of data had been collected on the generalized psychobehavioral traits of these same populations (Shuey). A number of works on the raciology of civilization had indicated that some genetic correlation between "ethnicity" and culture may exist. On that basis, the patterns of civilization could be explained in terms of genetic metamorphoses, actuated by disease, "dysgenic" breeding, or through the mating of radically different populations (Hrdlicka).

By 1930, sufficient data had been collected and adequately analyzed for Dr Ales Hrdlicka, founder and lifelong chief editor

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of the *American Journal of Physical Anthropology,* to summarize the major facts as follows: "The mental differences between the races, numerous and in some cases important, elude thus far direct and precise specification or determination. Sensory differences exist, but their exact nature and degree remain to be established. There are, between the more distinct races at least, apparently substantial differences in the higher psychical processes, but they have not yet been precised; their study is much complicated by what are merely mental habits" (Hrdlicka, pp. 159-160).

Dr Hrdlicka (p. 169) goes on to chart, briefly, some of the more notable gross anatomical brain differences between Negroes and Whites: —

	Brain size	*Cerebral convolutions*
White	Medium to large.	Medium to rich and deep.
Black	Small to medium, rarely above.	Relatively simple and shallow.

And finally, Dr Hrdlicka (p. 181) takes note of the rise and fall of civilization: "In human history 'race' after 'race' has risen to power and cultural prominence, only sooner or later to go down before some stronger group. This up—linger—and down phenomenon has in fact up to recent times been the invariable rule. Its principle cause has often been believed to be 'race degeneracy' . . . If mentality be excluded, no such degeneracy in any instance can be detected by anthropologists."

As far as this writer is aware, no information has been adduced since 1930 to warrant a dismissal of Hrdlicka's findings. Quite the contrary, what evidence, produced since 1930, has, if anything, corroborated the earlier studies with that very precision and specification that Dr Hrdlicka desired.

On that basis, it must then be asked, what has changed? What has caused so many presumably otherwise honorable scientists to deny the existence of brain and psycho-behavioral differences between human races?

The data remain the same, the people remain basically the same. What has changed? It is the times that have changed. It is no longer fashionable to explain human phenomena, above the level of the neck and within the endocranium, in terms other than environment.

One looks to the past for explanations on the origin of this prevailing situation. One need not look far. The excesses of

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Nazi neurotics between 1933 and 1945 have verbally immobilized a sufficient number of well-meaning individuals, to string a veritable iron rope of egalitarianism across our land and around the neck of the world.

The perversion of the truth is, of course, no reason to abandon it. From race X being all this way and race Y all that way, to all races being all things to the same degree at the same time. . . . From the concept of the master race to the notion that no races exist at all. . . . One sees a giant pendulum of attitudes swinging to and fro, with man's fate hanging in the balance. It seems that perversion only begets perversion.

That Adolf Hitler and his coterie of fanatical idealogues and murderers should stimulate opposition in all normal people is fine, defensible, and within the Western code and tradition, morally incumbent. That a backlash by a clique should occur (Franz Boaz, Otto Klineberg, Ruth Benedict, Margaret Mead, and so on) is understandable (Putnam).

That passion should receive judicial or statutory codification in at least two countries in a single century (such as the Nüremberg laws in Germany and the various Supreme Court desegregation decisions in the United States) merely emphasizes the need to remain calm. A failure to do so will only serve to carry out the work of the Hitlerian fanatics and the Boazian school which has leaned far over to unqualified egalitarianism.

How ironic that the worst of enemies should have so much of importance in common. How sad that their emotionalism has been allowed to replace the dispassionate appraisal of facts, in so much of anthropology, psychology, and biology. How tragic, that through fear and intimidation (as in Hitler's Germany) so many have remained so silent, so long. With these facts in mind, let us turn now to a consideration of Dr Phillip V. Tobias's article " Brain Size, Grey Matter and Race—Fact or Fiction? " as it appeared in the January-March 1970 issue of the *American Journal of Physical Anthropology.*

" The theme of this paper—Brain Size, Grey Matter and Race—Fact or Fiction?" writes Dr Tobias (p. 3) " is based upon the often repeated claims that the races of man have been shown to differ in quantity of brain-substance and, especially, of grey matter in the cerebral cortex. . . . It will be my aim to examine the evidence of such claims: to try to dissect fact from fiction. Finally, we shall be led to regard somewhat more critically the claims made about grey matter in men today."

While it will not be possible or necessary to examine every point made by Dr Tobias in his article, we shall try to examine what this writer feels to be the major points or thrust of his work. We shall attempt to find out how well Dr Tobias dissected

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fact from fiction, how critical his claims "about grey matter in men" are.

Considering the fact that significant racial differences have been demonstrated (Kuttner) in a nearly endless number of anatomical, biochemical, and soft external morphological characters, it is difficult to understand how Dr Tobias expects his readers to believe that such variation disappears as soon as one comes to deal with the subcutaneous traits above the level of the neck.

The fact that various psycho-behavioral traits (such as cognitive intellect, co-operation, physicaal vigor, and so on) may have positive selective value in most cultures has no bearing on the preceding.

All people need beating hearts. Do all hearts beat at the same rate?

All people need air to breathe. Do they all require the same intake of oxygen?

All people need sunshine for bodily growth. Do they all require the same period of time outdoors?

All people need sleep to remain alert. Do they all require the same number of hours?

The variation in the preceding is, of course, substantial. The list can be extended almost indefinitely. Its bearing on man's cerebral evolution is obvious. One need only concede that variation in human beings exist and the basis of invariate racial constancy in cerebral development is invalidated and rendered untenable.

When it is also borne in mind that the external soft anatomical characters used by somatypists to distinguish races, and the neurological characters, which cerebral morphologists use, are both of ectodermal embryological origin, how can one rationally admit that substantial variation in the former occurred through time, while the latter remained constant?

Such a contrived and artificial theorem is, of course, irreconcilable with non-linear human descent on the one hand, and the relative *plasticity* of human traits during non-linear descent (which Tobias champions on pp. 12 and 19) in response to environmental variation, on the other. It is also at variance with the facts of human embryology.

To dismiss the development of cerebral racial differences is to either deny the branching course of human evolution or to ignore the great regional variation in environmental challenges over millennia. Of course neither position is tenable, so again, racial constancy in cerebral development is invalidated. Indeed it is by all reason rendered non-controversial.

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As previously noted, Dr Tobias presents data illustrating both the aforementioned occurrences and as such has completely contradicted and invalidated the implicit theme and only possible, though irrational basis, of his entire report.

With a host of "modern technological methods" to study the brain, including electron miscroscopy, spectromicrophotometry and cybernetic models, what could remain to be said about the "overall size of the brain?," Dr Tobias asks (p. 82). F. Clark Howell, writing for *Time Inc.*, answers: "Sheer size in brains is important for two reasons. Most obviously, a small brain simply cannot hold as many brain cells as a large one. Less obvious but more important is that the true quality of a brain must be measured by the complexity of the linkages between cells. Inasmuch as the complexity of the linkages goes up very rapidly as the brain gets larger, it is clear that a big brain can be a much more sophisticated instrument than a small one."

It may be noted, in addition, that the use to which brain cells are put, are of great importance also, and since a larger brain contains more cells, there exists a greater possible range of neurochemical or functional variation; hence a still more sophisticated organ and more sophisticated behavior.

In other words, other factors considered (sex, proportion of components, the amount of cortex such as is reflected in how marked the convolution pattern is, and so on), and considering fossil man in a phyletic series, the gross measure of brain size or weight is one of a number of reliable indices, useful and necessary, in assessing human evolution.

For the aforementioned and other reasons, much needs to be said on "so gross and crude a measure as overall brain size." It may be added, as Howell also emphasizes, that individual exceptions may occur, that is, big-brained idiots or small-brained geniuses; hence there are other neural factors (such as an exceptional arrangement of localized cortical neurons in idiot savants) to be considered, in rare, selected, and atypical individuals, not populations. This does no violence to normal and general population trends. And it is the population which is under consideration here.

SOME CLAIMS ABOUT RACE AND BRAIN POWER

In this part of his report Tobias contrasts brain weight comparisons with cranial capacity comparisons and concludes, apparently for the purposes of his report, that the former is a more reliable index of brain size (p. 5). (This writer personally questions this conclusion but the reasons need not be considered herein.)

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Tobias pointedly and conveniently eliminates from evaluation numerous studies on the cranial capacity of Blacks and Whites. By removing from discussion one of the major pieces of corroborating data that demonstrates the existence of racial differences in brain size, Tobias not only makes his task of discrediting observed differences easier, but creates a distorted and misleading picture of the volume and extent of the evidence. It may be added that the formulae exist to compute brain weight in grams, from cranial capacity and *vice versa* (Gates, Matthews) hence, one need not necessarily weigh the brain to obtain reasonably accurate assessment of its size.

SOME FACTS ON BRAIN WEIGHT

After a short historical review on racial studies in brain weight, Tobias cites the longest American Black series on record, the American Civil War material analyzed by Raymond Pearl. It is cited to the effect that the brains of unmixed Negroids are larger than those of Mullatoes. The figures in question are derived from a chart, which appeared originally in an article entitled "The Negro as a Soldier," by Dr S. B. Hunt and Surgeon Ira Russell, in an 1869 issue of the *Anthropological Review*.

The mean weight of the brain for various grades of Blacks, seriated on the basis of pigmentation, and reported in ounces avoirdupois, were converted by Dr Pearl into the more conventional gram measure and published in a 1934 issue of *Science* (pp. 431-434). The later figures were then cited by Tobias.

Dr Tobias failed to note, however, that the original mean brain weight (46.96 ounces avoirdupois) for the apparently unmixed Negroids, calculated by Hunt, was based on an incidence of 141 brains (which were subdivided into range subgroups), yielding a ratio of brain number to mean brain weight of 141 to 3333. However, as J. Barnard Davis (1869) showed in an appendage to the article, the actual number of brains in the Hunt-Russell chart was 139.

When Dr Pearl calculated the mean brain weight for this series he used the tabular figure of 139 for the number of unmixed Negroid brains and 46.96 for their mean. His median was based on the assumption that the mid-point in brain weight, for each range sub-group, could be taken as its median. By further assuming the average weight of the two missing brains (whose weight is unknown) to be 46.96 ounces each, Pearl, not unjustly, at least in his mind, eliminated them from computation.

In this way he absorbed an artificial decrease in the ratio of brain number to mean brain weight for the entire series (from 141 to 3333 to 139 to 3377) and an arithmetically consequent

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inflation in the weight of the series, producing a 1354.8 gram mean. His comparable median figure was, as previously explained, arrived at by the mid-point assumption.

In order to compute the average brain weight for this group on a 139 brain basis (which is the only reasonable way to do it since there are only 139 brains available) without causing the afore-demonstrated mathematical variances, and the invocation of unwarranted assumptions, one must equate for the 1.4 per cent decrement in brain number, as well as maintain the original and correct ratio between brain number and mean brain weight.

Equated for these factors, the mean brain weight for Hunt and Russell's pure Negroid Civil War soldiers is 46.28 ounces avoirdupois or 1311.9 grams. This compares with a mean brain weight of 1333.6 grams for the series of mixtures reported by Pearl.

In other words the unmixed Negro brains do not weigh more than the mean for the series of mixtures. Tobias's claim to the contrary is without basis in fact.

It appears to this writer that the original Hunt figures, taken under war conditions, went slightly awry, due to adding up the total weight for 141 brains, and dividing their weight by only 139. The latter number of brains were then put in their chart into frequency distribution sub-groups, and published. The consequent variance was, however, relatively small, amounting to about a 3 per cent increase in mean brain weight.

It may be mentioned that the Blacks in question are escaped slaves from the South who migrated North to fight for the Northern Army. Hence, selective migration and selection, in that they are escaped slaves, are factors to be considered in evaluating the absolute mean weights for these Negroid series (Hunt).

Tobias then turns to a consideration of what he terms the "biggest series of White or Caucasoid brains to have been weighed . . ." (p. 6), a series of 2752 Americans by F. W. and E. M. Appel.

What kind of series was this? I quote Appel and Appel from the second page of their article (p. 49): "No racial subdivision of this group of cases is possible. Diverse European stocks are undoubtedly represented and *possibly there is some admixture of non-European stock*. The patients came to *St Elizabeth's Hospital* from the civil and military services of the United States government. Presumably they came from all sections of America" (emphasis mine).

In other words this series cannot honestly be claimed as a White series in the usually accepted sense; not even Appel and Appel completely affirm a monoracial provenence for every

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brain. What kind of a hospital is St Elizabeth's? A mental hospital!

The legerdemain of injecting mental patient material into a discussion of mentally normal brain series is both inappropriate and unacceptable, since, by general definition, such individuals have deviant brains and mentalities to begin with.

Tobias (p. 6) then concludes, on the basis of the series of Appel and Appel, and Pearl, that Negro populations sampled in his paper have averages above and below the overall modern series (1344 grams).

As we have seen under close analysis, such is not the case. The series' overlaps which Tobias stresses, on the basis of data presented in his reports, are, in fact, non-existent.

BRAIN SIZE AND BODY SIZE

Tobias opens this section of his article with an examination of the relationship between brain weight and body weight. He surveys a representative sample of animals, and finds, for example, that house mice and porpoises have greater brain to body weight ratios than humans (p. 7). Tobias completely and inexcusably ignores brain structure and morphology in his evaluations.

In the case of the porpoise (Langworthy, and Morgan and Yakovlev), it is pertinent to note, that there is an almost complete absence of frontal cortex anterior to the genu, a very small frontal lobe, and little neocortex. Also, no animal in nature, to my knowledge, has as low a percentage of spinal cord in proportion to brain weight as humans (Latimer and Krompecher and Lipak, p. 117). This accounts for the high brain to body weight ratio of the house mouse.

Tobias then turns to an analysis of the relationship between stature and brain weight. He cites the studies of Span and Dustmann, Schreider, and Pakkenberg and Voigt, on the apparent positive correlations between stature and brain size (Tobias, pp. 6-7). None of these writers controlled for age, within their young adult and adult series, although studies show variations in brain size and stature during adult life (Pearl, 1905, pp. 13-61).

Raymond Pearl (1905, pp. 51-60), whom, curiously enough, Tobias cites in support of his thesis, showed statistically, that once a correction is made for age, the correlation between body height and brain size in humans is mathematically trivial.

The superficial correlations observed by several other writers are merely a result of sampling without control for growth and senescent functional variation in brain weight and stature.

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In Pearl's (1905, pp. 62-63) words, "without any question, I think, the correlation (between stature and brain weight) is to be regarded in its origin as a growth correlation, the relation between the two variables being practically fixed at the end of the growth period. After that time the relation undergoes no marked change throughout the remainder of life, since both stature and brain weight regress on age at not far from the same rate." Socio-economic factors may play a slight rôle since taller people and larger-brained people tend to be of the upper social classes.

In light of the fact that brain to body size comparisons in non-humans have generally been made between different genera or sub-families, a lack of correlation in man is not surprising. Similarly, since both body weight and stature, other factors being equal (sex, age, diet, endocrine balance), are a function of the size of the bones, and appendages, and muscles and their extension, all of which are of mesodermal embryonic origin, whereas brain size, which is a product of nervous system development, is of ectodermal origin, there is no embryological basis for anticipating brain and body size to be covariates. The fact that size, generally, and bodily components individually, are affected through hormonal action, does no violence to the preceding, since symmetry is genetically determined and specially, racially, and individually specific (again, the aforementioned factors being equal).

As a functional exhibition of the aforementioned fact, we may note, that the African pygmies, with mean brain capacity of 1428 cubic centimeters (Twieselman), have as large or larger a brain that the full-sized Negro, whose mean cranial capacity has been placed by Karl Pearson at 1350 cubic centimeters (Benington).

Indeed, if Tobias's reasoning is followed to its logical conclusion, one could never have valid brain weight comparisons between Nilotic Negroes and pygmy Blacks, since there is no overlap at all in regard to stature between the two groups.

In point of fact, while it is generally agreed that there has been no increase in human brain size, at least in the last 40,000 years, stature increase in various areas are often noted. These increases, which have been particularly pronounced in the last 50 years, are very well known and need not be documented or considered herein.

Tobias's conclusion (p. 9) that "if one does not allow for the varying average body sizes of different populations, one may be misled into making false statements about the existence of racial differences in average brain size among populations compared" is without factual foundation.

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Dr Tobias (p. 8) goes on to dismiss the findings on low Negro brain size by F. W. Vint, J. B. Davis (1869) and Raymond Pearl (1934) because they allegedly failed to take body height into account.

Pretending, for the sake of discussion, that body height is a significant factor in adult brain size, let us consider the following pieces of data. Dr Ernest Hooton tells us that the average white population has a "stature from 160 cm. to 169.6 cm." (pp. 539-540). He goes on to report that "In Africa, *great bodily height* (over 170 cm.) *is commoner than in any other continent.* It occurs especially in the *head waters of the Nile, in the eastern Lake Region,* and *among the Bantu of South Africa* (emphasis mine).

What are the facts? Dr Vint's series consisted entirely of Nilotic Negroes (15 per cent) who are not infrequently seven feet tall and are the tallest people in the world, and the tall East African Bantu speaking Negroes (85 per cent). Davis's brain weights were calculated from skull measurements, while Pearl's analysis was based entirely on individuals around 21 years of age (Hunt, p. 52). Hence, the recording of stature was unnecessary in all of these studies. Tobias goes on to state that "no height measurements were recorded . . . for any of the earlier cited studies."

I suggest that Dr Tobias read or reread, just for example, Robert Bennett Bean (1906 and 1917), T. Wingate Todd (1913). In these works he will find the recording or correction for stature. Negroid brain size and/or differential with Whites, in spite of comparing, in some cases, low class and criminal element Whites, with representative or often above social average Blacks, is clear and consistent with the data cited by Tobias.

Tobias's final conclusion (p. 9) in this section, that "on this basis alone [stature] all comparisons between Negro and White brain sizes to date are invalid" can unequivocally be labelled for what it is, *pure fiction.* It may also be noted that between sun up and sun down, you lose, on the average, 2.5 cm. in stature, which you regain during a night's sleep spent horizontally. Does Tobias expect his readers to believe that a concordant rise and fall and rise again (i.e. fluctuation) in brain size transpires during, and as a function of these events?! (Backman).

BRAIN SIZE AND AGE

Dr Tobias (pp. 9-10) shows his readers that brain size is dependant to a certain extent upon age, and cites European studies in which age is recorded. I have no quarrel with this part of his section. Dr Tobias however, goes on to state: "But this

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is not true of Negroid series, such as Vint on Kenya African brains. We simply do not know the age of his Negroid subjects. . . ."

This statement is not correct, as an inspection of the second table on page 217 of Vint's report shows. Here we find his subjects divided into seven age groups on the basis of age bracket and the mean weight in grams recorded for each sub-series. It is also to be noted that Negro brain weight remains relatively stable between 16 and 40 years of age. Dr Vint (p. 217) writes: "My post-mortem records suggested that the brain of the native youth is as heavy as that of the adult. Therefore, the average weight was calculated by age groups for those cases in which the age was known. . . ."

BRAIN SIZE AND NUTRITIONAL STATE

"Evidence has accumulated, especially in recent years, that undernutrition at critical stages during ontogeny of [humans and] experimental animals may lead to permanent impairment of the brain, including diminution of brain size," writes Tobias (p. 10).

It is very important to note that Dr Tobias opens this section of his article with a discussion of undernutrition or malnutrition. What data does he cite in support of his thesis? The following highly selective group of witnesses are called to testify: Jackson and Stewart; Dobbing and Widdowson; Davison and Dobbing; Dickerson and Dobbing; Dickerson and McCance; Dickerson and Walmsley; Guthrie and Brown; Zamenof van Marthens and Margolis; Chase, Lindsley and O'Brien; Eayrs and Horn; Kerple-Fronius; Fishman, Prensky and Dodge; Stoch and Smythe (1963 and 1967); the widely known study of Cravioto and Robles; Engel; Nelson (19559); Brown (1966); Baraitser and Evans; and Strom—concentration camp survivors! Tobias also mentions articles or works which cite some of the preceding studies in drawing their conclusions (Abelson; Schrimshaw and Gordon; and Eichenwald and Fry).

It will be seen from Tables I and II, as well as from the very titles of some of the works cited what the preceding studies share in common.

They do not meet the medical definition of malnutrition, which *Taber's Encyclopedic Medical Dictionary* defines as a "lack of necessary food substances in the body or improper absorption and distribution of them." They all fall within the medical perimeters of disease and/or starvation. "Starvation is the condition of being without food for a long time . . . the condition in which the supply of specific food or food accessory is *below*

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TABLE I

Mean statistical losses as a product of starving to death
(Moore, p. 45)

Subcutaneous fat loss	97%
Heart and brain weight loss	3%
Skeletal muscles loss	33%
Liver and spleen loss	50-60%
Kidneys loss	25%
Body weight loss	25-40%

(It may be noted from the preceding table that a 3 per cent loss in brain weight stands in marked contrast and does not notably reduce the 8 per cent to 12 per cent differences between Blacks and Whites, reported by Tobias (p. 8)).

TABLE II

Stoch and Smythe	Marasmus disease, also starvation; mean weight of abnormals 5.21 kg. 2.50 percentile controls 8.16 kg. 10th percentile (pp. 1027-31 and 546-552).
Dobbing and Widdowson	Starvation - brains normal; extreme body weight loss as in preceding studies (pp. 352-366).
Dickerson and McCance	Starvation-body weight loss in suckling pig at one year of age; normal pigs weighed 150 kg.—starved pigs 5.4 kg. (p. 398).
Dickerson and Walmsley	One-third body weight loss; starvation; brains normal (pp. 897-900).
Baraitser and Evans	Acute marasmus, the same 20 children as in Stocke and Smythe studies (pp. 56-58).
Cravioto and Robles	Kwashiorkor disease (pp. 449-464).
Guthrie and Brown	Body loss at starvation level (pp. 419-421).
Chase, Lindsley and O'Brian	Starvation; same reasons as the preceding study (pp. 554-555).

minimum bodily requirement (e.g. protein starvation)" (Taber, p. 572). Kwashiorkor, the condition in which many of the individuals in the studies cited by Tobias found themselves, is "A *disease* resulting from a *deficiency of protein in infancy* or early childhood." (Emphasis mine.) (Taber, p. K10.)

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For Tobias to open this section of his report with a discussion of malnutrition, only to cite evidence bearing on starvation, is clearly irrelevant and misleading. To obfuscate matters still further, by injecting a second variable irrelevant to the opening discussion, in this instance disease, suggests either a lack of understanding of the issue, or a lack of evidence to support his contention. In either case his material is inappropriate. Indeed, disease can certainly not be used interchangeably with nutritional status regardless of causation, since diseases of the central nervous system can attack and inflict damage on the brain, independently of nutritional circumstance. Finally, one cannot rationally jump from starvation or disease, on the one hand, and claim that because brain damage may result in some of the former cases, that retardation, comparable or otherwise, will result from the latter. If this reasoning were followed through, we would have to fear for our health because of a slightly below recommended daily allowances diet.

Since starvation is not the norm in Africa (or any other continent) it cannot be invoked as a causal factor in Negro brain and mental test development. In point of fact, brain and mental test studies have almost invariably been carried out in and around areas of European contact, such as South Africa, where Kwashiorkor, with a frequency of affliction of about 1 in 1000 native Blacks (Weyl, p. 231), can only, if at all to an appreciable degree, serve to explain the cerebral or mental level of rare *individuals, not populations.* It is also to be noted, as Cravioto and Robles have shown (pp. 449-464), that neonatal brain damage and Kwashiorkor causes a retardation in psychomotor development. The facts are that infant Negro populations sampled throughout Black Africa (Weyl, pp. 221-231), and in America (Davenport and Steggarda) exhibit developmental maturative milestones at age periods earlier than White infants, regardless of weaning or not weaning. These facts are wholly inconsistent and inimicable to malnutrition, let alone neural disease and starvation.

In connection with diseases that attack the central nervous system, it is often stated that spirochoetal disease may play a rôle in brain structure and mental test score level. However, as Dr J. S. Bolton (pp. 386-387 and 415-434) of Leeds University, England, has shown, spirochoeta disease causes no microscopically visible desiccation, or reduction of brain tissue in normal or sane individuals, only possibly affecting the brains of abnormal individuals already possessing degenerated encephalons, as a secondary factor. This is analogous to tinder responding to flame. Where there is no tinder, there can be no response. It is

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also to be noted that Whites so afflicted score markedly higher than Negroes on I.Q. tests (Shuey).

In studying malnutrition, we must divide it into two major categories, prenatal and postnatal. Because the developing fetus has not reached that stage of development where it is a biologically full and "self-contained" human being, a pregnant woman's sub-nutritional status might play havoc with reproductive processes. On the other hand, the newborn infant, child, adolescent, or adult are now biologically "self-contained" units or beings, equipped to face the world. For these and other reasons, it is advisable or necessary to study the effects of malnutrition on pre- and postnatal levels, separately. Let us first consider prenatal malnutrition.

Because of the bio-developmentally precarious position of the fetus, is it reasonable to expect that a pregnant woman's deficient diet will cause deleterious effects on the brain and bodily development of the unborn child, and if so, to what degree. Let us examine representative data.

In one study, first class breeding sows were given a diet deficient in vitamin A during early pregnancy. Every animal was born without eyeballs, and many other abnormalities (Hale, 1933).

A study which eliminated folic acid from the diets of pregnant rats produced many harmful results. When this nutrient was eliminated from the diet of the female rats on the ninth day of conception, the fetuses failed to develop and were resorbed. When omission was initiated on the eleventh day, 95 per cent of the rats had abnormalities (M. M. Nelson, 1955, and Sling).

Development in the heart and blood vessels were striking, numerous skeletal deformities were present, as were brain deformities. Some animals were born with brains outside their heads, while other animals did not have heads.

In a study (Burke) carried out by the Harvard School of Public Health, the diets of middle class human mothers during pregnancy were classed as good, fair, and poor. There were 216 infants born, who were individually rated. Thirty-three were rated poor, of these all were deformed, premature, stillborn, died in the first couple of days after birth, or were physically stunted. Of these cases, 26 came from mothers with poor diets, six whose mothers had fair diets, and only one whose mother was rated as having a good diet. This last case may well be due to an inherited familial genetic defect, while the others are clearly due to malnutrition, exhausted germ plasm, or likely both.

In connection with abnormal reproduction, we may note that

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prematurely born Black children, matched for weight with White children, performed significantly less well on mental tests at three and five years of age than the latter. It is also to be noted that prematurely born children are usually of normal intelligence (Cook).

For every 100,000 conceptions there are 7000 pathological embryos and 615 terata (monsters). Approximately one-third of all of these, or 2 per cent of all conceptions, are well-formed terata. Of each 100 terata, 75 are aborted in the early months of pregnancy, and 25 go on to term. On this basis there are about 0.6 per cent of all births at term which result in malformation (Moore, pp. 698-699).

Another study, this time on 17 normal rats, found that, when the amino acid, tryptophane, was eliminated from the diets of nine pregnant rats, none of them produced a litter of young, while all eight of the mothers on normal diets produced. All of the unsuccessful fetuses were resorbed within a week of conception. Nature simply gave up because there was not enough tryptophane to allow normal development (Albanese, Randall and Holt).

What do all of the preceding studies on pre-natal undernutrition show? They show, rather grossly, that without exception, whether in humans or non-humans, when a dietary deficiency is present, the damage, cerebral and otherwise, is unmistakably not subtle. They also show that there is, in terms of frequency, not a statistically significant number of newborn individuals who survive pre-natal malnutrition, notably physically, to affect the living population. In other words, nature has a protective mechanism to insure the fitness of the living group and race. We have seen, that as a rule, only the normal survive. The studies also show that a good diet, as a rule, is a prerequisite for normal reproduction. This must be made clear to prospective parents.

Let us turn now to a consideration of the effects of malnutrition when it occurs during postnatal development.

Brain growth is a matter of protein synthesis or anabolism. What happens in the body during protein or general malnutrition? To begin with Kimber, Grey and Stockpole (p.479) report: "The blood contains amino acids at all times; *fasting does not free the tissues from them, neither does a high protein diet result in any great increase* in the blood or tissues. *Amino acids are considered intermediary products in the building up and breaking down of body protein*" (emphasis mine).

The Textbook of Pathology (Moore, pp. 6-7) continues: "Within a few hours after the loss of from 20 to 30 grams of plasma protein, the volume of the blood is restored by the *with-*

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drawal of fluid from the tissues, and *within six to twelve hours the protein concentration of the blood has been restored to normal.* This indicates that *there is a storage of protein* in many parts of the body which *can be drawn upon for emergency use.* A part of this protein is in labile form and may be thrown into the circulation within a few hours, while the other part is more stable and cannot be used immediately (so it can then be requisitioned over extended periods). Different organs store protein in varying amounts, so far as can be determined by chemical analysis this stored protein is identical with cytoplasmic protein" (emphasis mine). When protein stores are being depleted, this is called *tissue wasting.*

What occurs and precedes the complete utilization of tissue protein and the wasting of tissue (negative nitrogen balance)? First, *stored carbohydrate is utilized* (Moore, pp. 44-45); at this point there is a fall in the urinary excretion of nitrogen, followed in three to four days by an increase, reflective of the oxidation of stored protein. By the fifth day there is another decline to a constant level of the order of 6 grams a day, until a premortal increase signals *utilization of tissue protein.* Urinary urea first rises but then progressively decreases. *The burning of fats after the depletion of the carbohydrates* causes incomplete oxidation, the appearance of acetone bodies and ammonia in the urine, and the chemical changes of acidosis, all caused by the commencement of faulty metabolism. The *abnormal muscular metabolism* causes a decrease of creatinine (a non-protein blood constituent) and an increase of creatine (a colorless crystalline substance) in the urine. What do the latter activities reflect? They no longer may be termed signs of malnutrition, by definition, they are again the criteria of starvation.

In other words, before utilization of the bodily store of protein, carbohydrate and fat utilization transpires. These are signs of malnutrition. During this period, protein levels remain constant because of the protein stores, and utilization cycles of bodily nutrients discussed.

Since both normal metabolism and the building material of the brain, protein, remain at normal and constant levels during malnutrition and only fall to sub-necessity or depleted levels during starvation, how can malnutrition possibly cause brain tissue damage, desiccation or wasting? The question is of course rhetorical and the answer patent.

Conversely, since death results or normal nutrition is restored, soon after abnormal metabolism commences and after protein reserves are being utilized and depleted (i.e. starving to death), and only after carbohydrate and fat stores are exhausted (during which time, as we have seen, we can expect a marked reduction

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in subcutaneous fat, among other changes), we cannot expect a great loss, if any, in brain weight. On the other hand, tissues and organs whose size is less fully dependent on protein intake, can be expected to lose more weight than the brain. As has been shown in Tables I and II, this is precisely the case.

Kerple-Fronius, whom Tobias cited in favor of his thesis suggesting deleterious effects on brain growth from malnutrition on a three-month-old infant, was enthusiastic to emphasize that the starved (50 per cent body weight loss) child "went on growing" with no evidence of cessation, and that the estimated 10 to 20 per cent decrement in brain weight which he noted, was not to be construed as an immutable shortcoming to be wholly unalleviated by further growth in ontogeny (Kerple-Fronius, p. 10). The very title of the book, *Somatic Stability in the Newly-born,* was in fact suggestive of the findings, which Tobias found necessary to misrepresent. Indeed, Dobbing and Widdowson, whom Tobias (p. 10) cites erroneously or incorrectly to the effect that malnutrition causes brain shrinkage or damage, actually found, in spite of starvation (a loss of one-third body weight, p. 358) that (p. 364) "*complete rehabilitation* was achieved during the latter period of nutrition" (emphasis mine). And (p. 365) "The capacity for synthetic processes in the brain, which have been held back by undernutrition, may have considerable significance for malnutrition in human children."

Similarly, Dickerson and Walmsley found (p. 906), and also in spite of one-third body weight loss (in other words, starvation), "no significant difference between either the concentration or absolute amount of DNA-P in the brains of the experimental animals and their controls." The amount of phospholipid, short of starvation, is almost invariate, regardless of diet. Each of the tissues contains a characteristic amount of phospholipid (Moore, p. 53). The approximate content of the various organs of the beef per 100 grams of fresh tissue are: brain 4.58, liver 3.06, pancreas 1.86, kidney 1.62, lung 1.25, heart 1.64, and skeletal muscle 0.42 to 1.06. And commensurately, Donaldson found malnutrition to have no effect on myelination.

The decisive factor in brain weight loss appears to be whether or not the starved individual dies without prior rehabilitation. And even then, as we have seen, the brain weight or size loss is quite small; so small in fact as to suggest mere dehydration as the basic causal factor. It may be added that Brown (pp. 514-519) showed that Negroid children, who fall by definition within the medical perimeters of malnutrition, have "normal sized brains," whereas children suffering from marasmus, a *wasting disease* (Taber, p. M9) and kwashiorkor, have brains somewhat below normal (Brown).

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Corroborating the biological data, numerous studies (Hoefer and Hardy; Dowd; Hornell, Woodward and Gates; Nichols; Peterson); on cognitive psychometric intelligence have failed to uncover any demonstrable or permanent difference in test scores between malnourished and non-malnourished born and reared children, although notable differences in body size, strength and fatigue have been reported.

The facts were summed up well, and long ago, by Dowd of Bellevue Children's Clinic. The report concludes as follows: "In general, our experience has agreed with other investigators that undernourished children distribute themselves in tests of intelligence similarly to normal children; that children of high general intelligence gain more nutritional care than those of lower scores. . . ."

What does the United States Government have to say on nutrition? The critical dietary factor, as the U.S. Department of Agriculture (1971), the National Academy of Sciences (1968), the American Food and Drug Administration (1941), the Food and Nutrition Board of the National Research Council (1941) and the Federal Registrar (1957) emphasize, is not between recommended daily requirements and below requirements, but between a diet consisting of *minimum dietary requirements and a diet below minimum dietary requirements.*

And, once again, a diet below minimum dietary requirements is both starvation, as well as a disease progenitor.

As the Department of Agriculture (1971, p. 41) succinctly put it: "Note—the *Recommended Daily Dietary Allowances should not be confused with Minimum Daily Requirements.* The Recommended Dietary Allowances are amounts of nutrients recommended by the Food and Nutrition Board of National Research Council and are considered adequate for maintenance of good nutrition in healthy persons in the United States. The allowances are revised from time to time in accordance with newer knowledge of nutritional needs. The Minimum Daily Requirements are the amounts of selected nutrients that have been established by the Food and Drug Administration as standards for labeling purposes of foods and pharmaceutical preparations for special dietary uses. These are the amounts regarded as necessary in the diet for the prevention of deficiency diseases and generally are less than the Recommended Dietary Allowances" (emphasis mine).

And I wish to add once again, deficiency disease is a concomitant of starvation. In accordance with all of the data to my knowledge, The Committee on Medical Nutrition, National Research Council (Taber, p. M6), reports a long list of physical maladies, actuated by (infant, child, adolescent, and adult)

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malnutrition. Lacking in their list is a report on brain and mental test score changes or damage.

There is no apparent excuse for flagrantly exaggerating the disabilities brought about by postnatal malnutrition and needlessly frightening parents, who may not have the funds to provide a completely well-balanced diet for their growing children. It is from a neural standpoint, prenatal nutrition to which prospective parents may be concerned.

As we have learned in our critical examination of prenatal malnutrition, nature protects and insures the survival of the living population, by eliminating to a nearly absolute degree, defective offspring who owe their condition to nutritional circumstance. This was to be expected, in light of the fact that an abundance of highly mentally and physically unfit individuals would tend to inhibit the survival of the living population and race during the course of human evolution. And I think we can be reasonably certain that during man's evolutionary development he has found himself under conditions of malnutrition many a time.

Similarly, in dealing with postnatal malnutrition, natural selection could have been expected over the millennia to increase the frequency and stabilize those genes which ensure normal ontogenic development and neuroanatomic-physiological plasticity and durability in times of nutritional stress (inadequacy), thereby, again, preserving the population and the race.

It should be remembered, in connection with the differential methods employed by nature, in pre-natal undernutrition on the one hand, and postnatal malnutrition on the other, that there is rarely a case where a single method of alleviation, in any field, has absolute primacy in terms of benefit under all conditions.

In summation, we have seen that prenatal malnutrition has, from a frequency standpoint, no significant effect on the brains and bodies of living populations, while postnatal malnutrition has no permanent demonstrable effect, and not even starvation can cause marked or necessarily irreparable damage to the human brain, mind and body. It is also to be noted that there is no overall reduction in the size of the Negro brain, such as occurs in death through starvation. The reduction is due essentially to lower height of the brain (Vint), a condition as such, which is basically phylogenetic in origin, as Franz Weidenreich has shown. It is also pertinent to note that with the exception of one minor brain study, Tobias does not even suggest, and for obvious reasons, that Negroid series such as Vint's, Bean's or Pearl's contained other than apparently normal brains.

Finally, it is both significant and instructive to inform the reader that many White people may be malnourished also,

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despite Tobias's one-sided and misleading account to the contrary. For example, the American Medical Association (1942) finds that the majority of Americans are malnourished, and furthermore, pellagra, a deficiency disease with mental symptoms and CNS wasting, which is caused by a failure of the body to absorb niacin and a lack of protein containing tryptophane (remember the rats?), is or until recently was, endemic among the intelligent people of the white South (Moore, p. 710). This latter case is due to dietary tradition, not poverty.

Hence, in illness, as in malnutrition, we are dealing with a two-sided coin: one side Black, the other side White.

In closing this section, it can be stated that the malnourished and allegedly malnourished conditions that some living peoples find themselves in, has no demonstrable effect on the brain or measured cognitive intelligence. Tobias's suggestions to the opposite effect may be rejected.

BRAIN SIZE AND THE INFLUENCE OF NON-NUTRITIONAL ENVIRONMENT

In this section, Tobias (p. 12) investigates the possible effects of physical environment on brain size and structure. He cites the studies of Diamond (1964 and 1966), who, using both bright and dull strains of rats, found a deeper visual cortex and higher glia-neuron ratio in that area of the brain in rats raised in a normal environ than in rats raised in total isolation. The differences were not overwhelming, being about 5 per cent.

Tobias fails to mention Diamond's article in *Science* (1966), where three times greater differences in acetylcholinesterose activity were produced by selective breeding than by different environments (p. 10). It is also instructive to note that the bright rats showed greater response to the improvement of their environs than the dull rats.

It may also be mentioned that Dr Vint (p. 220-221) found the Negroid to have a deeper visual cortex than the European. This, the reader will remember, is the very cerebral area where Diamond's rich environment rats showed the greatest and really notable gains!

This writer must also confess ignorance as to the possible relevance of rats raised in isolation, to the sub-standard or allegedly sub-standard human condition. And perhaps still more important, after commenting on the effects of enriched experience on cortical weight, depth and total acetylcholinsterose activity, Diamond (1964, p. 618) states, "In the rest of the brain, acetylcholinesterose activity also increases, even though *tissue weight decreases with experience.* These changes have been produced consistently in many replications, they have

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appeared in each line of rats tested to date, and they are found in adult as well as young animals" (emphasis mine). In other words, in summing up, the Diamond and Vint data support the opposite of Tobias's suppositions!

Tobias goes on to cite studies showing that various species of animals raised in open rural environments have larger brains, on the order of 15 to 25 per cent, than animals raised in domesticated urban environments (Poliakova). In light of the fact that most African and American Negroids live under open rural conditions, as opposed to the usually constrained urban life of the White person, it is clear, using this criteria, that the Black is reared under conditions for maximum or optimal neural development, while the White person is at a decided disadvantage. As such, the conditions which most Blacks and Whites are living under *can only serve to attenuate, and quite strongly,* observed differences in brain weight and structure between the two groups.

BRAIN SIZE AND SOURCE OF SAMPLE

The apparent major point of this section of Tobias's article is to show the benefits of forensic medical brain material, which Tobias describes as obtained from individuals who were received "following sudden death without prior disease" (p. 13).

The apparent implications are that we would have a series more representative of civilian populations than regular hospital material, and that comparisons between forensic White series and non-forensic Black series are unfair.

To read Tobias's statements, one might gain the impression that normal healthy people were walking down the street, only suddenly to drop dead without prior warning.

Such hospital material includes individuals who have, for example, died, often at a relatively young age, of cardiac failure, strokes, execution for crimes, along with a number of people who have died as the result of accidents. Is the reader actually to believe that people who die at a young age of strokes and heart attacks have been previously normal? As doctors will tell their patients, the warning signs were there long before the end, they were merely not heeded or were ignored.

Since representative humans do not die young from such factors, or they are executed, or die from accidents, we cannot accept forensic medical material as any more representative of civilian populations than regular hospital material. In point of fact, the brain weight (1470.6 grams) for White Civil War soldiers (Hunt) is directly comparable to that of the forensic series of Mateiegka (Tobias, p. 13)—1455 grams—and Pakkenburg and Voigt (1450 grams).

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With the exception of mental hospital material, no difference of significance has been shown to exist between brain size or brain weight and hospital type. Public hospitals may, as Tobias suggests, tend to reflect the lower social strata, thereby yielding lower brain weights. But this, again, applies to both Blacks and Whites, and hence is eliminated as a factor of significance when evaluating racial brain studies. As a matter of fact, if Blacks almost invariably leave their bodies to public hospitals, while "well to do" Whites tend to have their bodies taken care of at private conveniences (as Tobias states), this would tend to remove the more selective Whites from brain series, while leaving a representative, and in comparison with the Whites, highly selective, Black brain series. This would tend to attenuate possible race differences in the brain. Thus Tobias's suggestion that selectivity in terms of socio-occupational differentiation might increase, or even explain observed brain weight differences between Blacks and Whites, is not only, by his own data, devoid of fact, but devoid of logic.

BRAIN SIZE AND OCCUPATION GROUPS

Tobias (pp. 13-14) presents data showing the variation of brain size with social class for Europeans. I have no argument with his data. However, it must be noted, since the great bulk of Negroes are of lower social strata, such a category is representative of their status. On the other hand, a middle class series would be representative of Whites. In other words, one must also consider racial variation in the distribution curve of social status. To do otherwise would tend to compare (as has been done) less than representative Whites with rather selective Negroes.

BRAIN SIZE AND THE CAUSE OF DEATH

In this section Tobias (pp. 14-15) suggests that various disease and non-disease categories of death may significantly affect the weight of Negro brain series. He again ignores the fact that White brain series are privy to the same conditions. Dr Tobias also fails to inform his readers of the following pertinent notes: (1) Since all brain series consist of individuals who have died from a variety of causes and, since accidental deaths—roughly 1 in 750 (Appel and Appel, p. 236)—do not account for a sufficient number of cases significantly to affect the mean of the series, this section of Tobias's report is only of passing interest, and not in any way explanatory of observed Negro-White differences in brain weight. (2) It is also to be emphasized that individuals who die accidentally or when executed, tend to

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be a little younger than disease-ridden individuals at the time of death. (3) And, finally, there may be age group differences between the various disease categories themselves, which tend to affect slightly their mean brain weights.

Although for reasons already given, the absolute weights of the Appel and Appel series is unacceptable, I think it is quite appropriate to cite the feeling of the two workers on the slight brain weight differences between the various disease and non-disease groups. (Since Tobias, as has been shown, cited their data in an attempt to explain observed Black and White brain weight differences.) Commenting on the significance of the relative, not the absolute, brain weight figures, Appel and Appel (p. 240) write, "It is very doubtful whether these relative differences should be construed to have any precise significance. We cannot take them that way."

Although the degree of intravital swelling tends to vary slightly with the nature of the disease, pointing to real differences, the actual variations in brain weight are not great, as Appel and Appel have emphasized, and cannot serve to explain notably, if at all, observed Black-White levels or differences in brain weight.

Finally, it is to be noted that individuals who fall prey to various diseases are likely to be less well-endowed genetically, at least insofar as their defense systems are concerned, than individuals who die through accidents. Then too, human mean fitness is likely to vary slightly with each category of disease, with both Blacks and Whites affected—and, therefore, there is no discrimination.

BRAIN SIZE AND LAPSE OF TIME AFTER DEATH

In this section we find Tobias (p. 15) contrasting the weights of deranged brains weighed immediately after death with deranged brains weighed hours and days after death. Tobias again cites the inappropriate and—even by Tobias's standards—defective study of Appel and Appel. Inappropriate because, as previously explained, it is a mental hospital series. Defective, by Tobias's criteria, because the writers do not know whether or not their brains were stripped of their meninges, drained of cerebrospinal fluid or weighed fresh.

Through the omission of explanatory statements by Appel and Appel, Tobias makes it appear that the time that elapses between death and removing the brain from the body causes a steady rise in weight, and is a critical factor to be taken into consideration when evaluating the mean weight of the brain in different normal series.

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What is the explanation for the phenomenon of rising brain weight post mortem? Appel and Appel (pp. 68 and 247) write: "The mean number of hours post mortem was found to increase as the brain weight increased, *and the mean age at death was found to decrease; to this extent our previous findings were verified* . . . but neither the change in time of 7.96 hours post mortem nor the change in age of 9.69 years *nor both together could account for such a change in weight,*" and the pivotal cause of "the enlargement of the brain seemed to be accounted for, at least in part, by this *internal traumatic hydrocephalus*" (emphasis mine).

What possible relevance do the preceding findings have, for example, to Vint's series of normals equated for age (pp. 216-217), or to Pakkenburg and Voigt's White series (pp. 217-300), which was equated for age and consisted of normal brains? That an appreciable expansion in brain tissue and size, in normal brains is impossible, was well demonstrated by Weed (1922 and 1933) many years before the study of Appel and Appel. Weed's work, incidentally, was cited with approval by Appel and Appel on page 245 of the 1942 article cited by Tobias.

Finally, Dr Tobias suggests that the "temperature the cadavers are kept between death and the removal of brains may affect the rate of chemical change, and so increase in weight after death."

Since White brain series usually come from cool or cold regions (such as North Europe), conditions which curb molecular activity (see "Grey Mater" section) and hence is inconsistent with a rise in brain weight, and since African Negroid series usually come from hot areas, conditions which favor increased molecular activity in the brain, and hence, if anything, a rise in weight or total size, such conditions can only serve to attenuate White-Negro brain weight differences. It will be noted that Tobias presents no evidence that such climatically functional post mortem neurochemical activity exists. It is also interesting to observe the statements of Appel and Appel (p. 49) on their samples: "Weights are available for several organs besides the brain, particularly the heart and spleen, the kidneys, and the liver. Unfortunately, neither the weight of the body nor any of its dimensions are given."

Again, if stature is such an important item in brain weight comparisons, how could Tobias honestly, rationally or consistently present this study as a legitimate reflector of the effects of "brain size and the cause of death" and "brain size and the lapse of time after death," or as an indicator of low mean White brain weight in "some facts on brain weight."

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BRAIN SIZE AND THE TREATMENT OF THE BRAIN AFTER DEATH

The technical procedures in brain weighing are reviewed by Tobias (pp. 15-16) in this area of his article. In his first point, Dr Tobias emphasizes possible significant differences in brain weight resulting from severing the spinal cord at "some little distance" variance at or around the point of pyramidal decussation.

Dr Tobias fails to inform his readers that the *entire* spinal cord constitutes only 2 per cent of the weight of the brain (Taber, p. B41), so patently, not even a six inch variance in the level of severing could invalidate the 8 to 12 per cent mean differences reported between Black and White series by Tobias. In fact, even if the entire weight of the cord was included or excluded, this would not materially affect the mean differences.

Tobias then goes on to mention that "the complete drainage of cerebral spinal fluid from the fresh brains may change its weight from as much as 50 grams" and that the removal of the meninges can cause about a 55 gram variance.

The preceding data are interesting, but what relevance has it, for example, to Vint's series, which was weighed fresh, with the ventricles full and the meninges intact? (Vint, pp. 216-217.)

It is also to be noted that nowhere in his article does Dr Tobias criticize a single brain study by name on these grounds, and for obvious reasons.

ON THE MEANINGLESSNESS OF MOST STUDIES ON BRAIN WEIGHT

Little need be said in this section of Tobias's report (p. 16) because it is only a synopsis of previous sections.

We have already seen what is correct and what is incorrect; what is relevant and what is not; what Dr Tobias supported by evidence and what he did not. Tobias's conclusion that "it is totally invalid at this state of our knowledge to cite the alleged small brain weight of Negroes as physical basis for differences in intelligence and behavior" is totally unwarranted, so far as the data he cited are concerned.

WHAT DO DIFFERENCES IN BRAIN SIZE MEAN?

Tobias says (p. 16): "Enormous variations in brain size may occur within the limits of normal functioning. Even well-known men may vary considerably."

With these facts in mind, Tobias informs us of small-brained geniuses such as Walter Whitman and Antole France and large-brained geniuses such as Daniel Webster and Ivan Turgenev.

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He fails to inform us, however, to take but one example, that Antole France had an unusually great amount of cerebral cortex, both absolutely and in proportion to his brain size, as reflected in the complexity of the convolutional patern of his cerebrum (Guillaume).

Tobias also informs us that a Melanesian woman had an endocranial capacity of only 790 cm., and states: "It was not the skull of a pathological microcephalic." He fails, however, to inform us whether or not the woman was normal or mentally retarded.

All of the small-brained geniuses Tobias lists (Franz, Gall and Whitman) expired at 70 years of age or more. Gambetta could hardly be called a genius. We must be careful to distinguish between those who truly achieve greatness and those who merely gain fame. Check any biographies or encyclopedias on the ages at death of Whitman, Gall and Franz. It seems to this writer that France's brain, at least, was less than impressive in weight to begin with, hence advancing age or senesence may have played less, if any, of a *discernable* rôle in his case than the norm. As previously mentioned, France possessed compensatory cerebral characters in spite of a small brain, to account, this writer feels, for his high intellect.

In considering brain weight and genius, Tobias breaks nearly all of his own rules (methods in brain weighing, source of sample, disease, nutritional and non-nutritional environment, type of hospital, cadaver temperatures, socio-economic strata, age, hours after death, stature, level of severance, and the utilization of cranial capacity size rather than brain weight) in presenting this material.

And finally, Tobias completely ignores statistical means or even frequency, in his presentation, and as such, this section of his report is *meaningless.*

BRAIN SIZE, EVOLUTION AND SURVIVAL

Dr Tobias begins this part of his article by belittling the importance of brain size as factor in survival. This writer cannot find a single fact adduced by Tobias in support of his thesis (pp. 18-20). We are told that Cro-Magnon and Neanderthal men had larger brains "on the average than their present-day European descendants" (p. 18).

On the Cro-Magnons, Hooton (p. 369) writes: "One also reads in many books in prehistoric archeology the statement that the Cro-Magnons had larger brains than modern man . . . this is nonsense. In the first place, the French method of measuring cranial capacity with lead shot yields much higher capacities than are correct." This applies to French Neanderthals also.

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In addition, endocranial casts, as is very well known, show relatively small frontal lobe in proportion to brain size in a number of Neanderthals. And finally, the small series all fall within the range of living North Europeans anyway. Tobias tells us we can have the wonders of life, although, pertinently, he does not state whether we can create or even maintain the good life with a " size 5.50 hat compared with a size 8.50. . . ."

Since Dr Tobias finds it necessary to dismiss a correlation between hat sizes and survival, I think it not unreasonable, on historical as well as biological grounds, to mention that Northerners, at the time of the American Civil War, had a larger mean hat size than the South, and defeated them. The latter had a larger hat size than the Mexicans and defeated them in the Mexican-American War. The larger-headed Barbarous Indians defeated the Toltecans and they were defeated by the people with the largest hats of all, American Whites, in the Indian Wars (Hunt, pp. 49-51) (Custer's last stand was a single battle, not a war). The slightly smaller brain size of Southerners is likely attributable to the endemicity of pellagra.

There may well be exceptions to the preceding cases, but unless Dr Tobias has a competent (more competent than his report) plan to end all wars, it seems at present a little safer to be on the big hat side. This is not to say that hat size is the only factor of importance in survival or winning wars, or that it is necessarily, taken alone, of any importance. All of the data on hat size is from the 1869 Hunt article that Tobias cited, and to the opposite effect of its author's intent. Its merits may be judged accordingly.

In closing his section, Tobias cites statements by G. Von Bonin and James Hunt on the fallibility of brain size alone as an accurate reflector of intellect. No one to my knowledge has ever maintained brain size alone is the only factor in dealing with intelligence or individuals.

It has been stated repeatedly in this paper, and the very people whom Tobias cites disapprovingly in his report (p. 4) have emphasized the importance of structural and morphological factors in assessing neural and intellectual development. The extent to which phyletic structural and morphological changes may accompany, or be a direct function of phylogenic increases in absolute brain size, need not concern us here. Why Tobias habitually discusses topics irrelevant, as answers, to statements he cites unfavorably, is the matter of concern. But let us now turn to an investigation of that mystical cortical grey matter.

THE AMOUNT OF GREY MATTER

In 1934, Vint published data on the thickness of the Negroid

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cortex. He found it to be 14.8 per cent thinner in the areas examined than the corresponding areas in the European brain (p. 220).

Tobias (p. 21) quotes Donald A. Swan as follows: ". . . Dr F. W. Vint's careful comparative studies of the cortical histology of the brains of European Whites and African Negroes."

He follows by quoting Carleton Putnam in these words: "In 1934 F. W. Vint, of the Medical Research Laboratory, Kenya, Africa, published the results of a comparative study of Negro and European brains in which he found that the supra-granular layer of the Negro cortex was about 15 per cent thinner than the Whites." Then Tobias states: "In both quotations, the wording conveys the impression that Vint had himself made a careful study of the European brains, as well as of Negro brains, whereas he had in fact studied only Negro cortices and compared his results *with those attained in one other study* (emphasis mine), by a different worker, namely Von Economo, in Europe. . . ."

The full statement of Swan (pp. 234-235) was as follows: "Dr Vint measured the thickness of several laminae of the cortex and compared the averages for each of the eight areas of the African brains with those given for the corresponding areas of European brains as measured by Von Economo."

Putnam (p. 52), though not mentioning Von Economo by name, states expressly: ". . . the European brains . . . were measured in Europe and the Negro brains . . . were measured in East Africa. . . ."

The reason Tobias apparently found it necessary to quote these two gentlemen out of context (like numerous other individuals cited in his report) rests no doubt on the same grounds that he claimed that Vint compared his findings with only a single other worker, Von Economo (Tobias, p. 20).

How were Vint's comparative assessments and findings made? In order to seek corroboration of the facts found in comparison with the European series of C. Von Economo, Dr Vint carried out an additional investigation. In this study he employed the methodology of J. S. Bolton, and used the figures in Bolton's series of cortices, as the reference standard. The findings obtained in this investigation showed no significant deviation from that using C. Von Economo's methods and figures for comparison. I quote Dr Vint in full on this matter (p. 222): "In an endeavour to check the figures in the foregoing table [Von Economo's table], measurements according to Bolton's method were carried out on the prefrontal, visuosensory, and visuo-psychic cortices (the ones available in Bolton's series). The results obtained by this method of measurement, on comparison with Bolton's

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figures, showed no significant deviation from the results already given."

It may also be mentioned that in this field, as in other fields, comparisons between studies by different writers are normal and commonplace, and not the unusual or unwarranted activity Tobias intimates.

Let us delve further into the unwarranted complaints and charges of Dr Phillip V. Tobias. Not content with the preceding misquotations, Dr Tobias goes still further. Continuing, he invokes methodological variation in sectioning and staining techniques, as possible reasons for the differences in cerebral cortical depth reported by Vint between Black and White brains. He again cites, quite inexplicably, the Diamond studies. He cites them, this time, to the effect that: " Diamond *et al.* measured the thickness of the rats' visual cortex by two different techniques: frozen sections stained by thionin, and celloidon sections stained by Windle's modified Nissl strain. The thickness measurements by the second method were only 60.50 per cent of those by the former."

To begin with, the actual figure reported by Diamond in the *Journal of Comparative Neurology* was 65 per cent, not 60.5 per cent (Diamond, 1966, p. 122).

Second, Diamond merely (1966, p. 122) mentioned in passing that the cortices " shrunk " in this, her replication study. Nowhere on that page, or elsewhere in the report, does Diamond state the specific cause of this " shrinkage."

Third, in the light of the fact that Diamond herself states, in the 1964 *Journal of Comparative Neurology* study (p. 133), that a solution of Indian ink was used in an attempt to demonstrate blood vessels which caused an abnormal expension of the vessels because of the *pressure of the needle,* the difference between the 1964 and 1966 micrometric figures may be attributed to this factor, which is an extra-histological preparation, abandoned, as Diamond explained (1964, p. 113) because of the abnormal *results.*

Fourth, celloidin does not cause shrinkage or alteration of cerebral cortical tissue (Bensley and Bensley, p. 51), nor does paraffin, unless one accidentally overheats it. (Preventing overheating is a matter of ordinary procedural carefulness.)

Finally, J. S. Bolton (pp. 29-30) in his experiments on the matter has shown that different staining and sectioning techniques are micrometrically irrelevant. Whether sectioned by freezing or in paraffin, or stained by modified Nissl or ammonia-iron-alum haemotoxylin, etc., comparison of the various micrometric figures obtained from the same or different cases, cut and stained by the same or different methods, failed to reveal any

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discernable differences in the characters or depth of the cortex.

Tobias's suggestion that variation in sectioning and/or staining technique may play a rôle in Vint's findings on the cortex is without factual foundation. It may be added that Bolton (p. 30) hypothesized that formalin, and Canada balsam mounting medium, may have played a rôle in the results. However, Bolton's hypothesis is rendered null and void because formaldehyde fixation solution has no significant effect on cortical depth and is functionally independent of other histological preparations (Kraus, Davison, and Weyl), and because Canada balsam (handled carefully to prevent acidity) and other non-alkalinizing mounting mediums, are neutral and do not alter the fixed or natural bulk of the tissues (Gurr, pp. 519-525). It appears that the uniform results in micrometry, regardless of *varying* staining and sectioning techniques, may be attributed to stability in molecular action in cerebral cortical tissues, under different chemical and climatic environs.

Still continuing on the same page, Dr Tobias (p. 21) explains to his readers why they cannot accept the validity of Dr Vint's finding that the Negroid cortex is on the average thinner than the White cortex. His objections may be briefly tabulated as follows: (1) Measuring technique; (2) Preservation technique; (3) Social class.

MEASURING TECHNIQUES

As Tobias notes, the cortex can be measured in several ways: "(i) on the flat external surface of the convolutions; (ii) at the lip of the adjacent sulcus or fissure; (iii) on the side of the fissure; (iv) at the bottom of the fissure."

Tobias continues (p. 21): "Von Economo pointed out that the thickness of the cortex on the summits of the convolutions was twice as great (as a rule) as the floor of the fissures, but *unfortunately he did not make clear whether he had followed Bolton's averaging method or some other approach* (emphasis mine). Thus, Vint makes clear that he is not exactly certain whether Von Economo's measurements on European brains were made in the area as were his (Vint's) measurements on Negro brains, namely only on the summit or crowns of the convolutions—"The boundaries are frequently wavy. . . . It would be easy for one worker consistently to measure from a different part of such a wavy boundary . . . and so introduce a systematic error into his results."

Vint states that he measured his Negroid cortices on the top flat external surface of the convolutions. Von Economo, as Tobias himself has pointed out, stated that the cortex is twice as thick there (as a rule) as on the floor of the fissures. If Von

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Economo had measured the European cortex on the floor of the fissure, which is generally only half as thick as the summit of the convolutions, or had given the averages of the two areas, or any other layers in between, and still gotten a thicker reading on the White cortex than Vint did on the Negro cortex, the intracortical findings would still be proportionately the same, and the White cortex might well be 29 to 30 per cent thicker than the Black cortex.

What are the facts? Von Economo published his figures in his "table of cortical thickness of the crowns of the gyri of the most important areas" (Vint, p. 22). There was no confusion in Vint's mind as to methodology to use (p. 220). Dr Vint (p. 225) writes: "The measurements of the native cortex, which are taken from the crown or flat external surface of the gyrus, are compared in Table I with the figures given by Von Economo for the corresponding areas of the European brain."

And, as we previously noted in this section, studies of Negroid cortices were also taken by Dr Vint, using J. S. Bolton's averaging method, and his findings were commensurate with the findings in the Von Economo comparison. The methodology employed by *Dr Vint was good, and, purposely in accord in all necessary details, with both the Von Economo and Bolton techniques, for comparative assessments and purposes.*

On page 22, Tobias mentions the various depth differences Vint observed in different areas of the Negroid cortex but pointedly fails to inform his readers that comparable variation is found in White brains, or any brains, as well as the fact that the cerebral cortex is notoriously variable from one neural region to another. Is this a clear case of Tobias seeking to mislead the naïve reader?

It may be added that measurements on the same brains, using the same measuring techniques, but by two different workers, yield virtually the same readings on the depth of the cortex (Diamond, *et al.*, 1966, p. 118).

Because of climatic conditions, Dr Tobias (p. 22) states that Vint was unable "to fix the brain by injection of the carotid arteries." Tobias leaves the impression to the reader unfamiliar with the Vint study that the brains were examined unfixed. The first portion of the Vint paragraph (p. 216) which Tobias so carefully omitted, reads as follows: "On removal of the brain from the body, it was weighed without removal of the meninges. It was then suspended by the basilar arteries in 10 per cent formal saline, care being taken that the brain did not touch the sides or the bottom of the container." He finished: "Owing to climatic and other local conditions, it was impossible to fix the brain by injection of the carotid arteries."

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In contrast to the all-permeating method employed by Vint, fixation by perfusion can be partially ineffective because shunts in the circulatory system can keep the perfusion fluid from reaching the cells to be fixed (Bensley and Bensley). The apparent implication of Tobias's quotation out of context, is brain shrinkage under African heat. This is, however, totally unacceptable when one considers the first law of thermodynamics, which states, briefly, that a definite amount of heat can be changed into a definite amount of mechanical energy, or work, while a definite amount of mechanical energy can be changed into a certain amount of heat.

In other words, a rise in temperature of an object means that the speed of its molecules has increased. Therefore, the molecules require more space in which to move, causing, *if anything,* expansion of the material which is heated.

This does not necessarily mean there would be a notable increase in the thickness of Vint's Negroid cortices or brains between removal from the body and weighing fresh, only that shrinkage was not possible. This would also attenuate possible racial differences.

It is also instructive to note that Vint found the visuo-sensory area of the infragranular cortex, as well as all but one area (the visuo-sensory cortex) of the lamina zonalis to be thicker than the corresponding areas of the European brain. How does one explain this overlap, with an environmental theorem?—Clearly one cannot.

SOCIAL CLASS

Finally, Tobias cites Vint to the effect that the Negro brains did not include any of the "so-called educated class" (p. 20). Here we find Tobias judging Negroid class strata by his own (i.e. Western) standard, something uncommon and surprising in an egalitarian-like work.

What are the facts? The Kikuyu, who comprise 48 per cent of Vint's series, are described by Ian Henderson (p. 12) as follows: "The Kikuyu are the Germans of tribal Africa. This tribe of 1,500,000 is noted for its devotion to education, its ability to work hard, and its intelligence. The tribal reserves, which are potentially fertile and most strategically placed, are close to *Nairobi* and the European settlement. *In the last 50 years the Kikuyu have had closer contact with European civilization than any other tribe in Kenya"* (emphasis mine).

George H. T. Kimble (p. 98), professor of history at Indiana University, emphasizes that "Western civilization has affected all African tribes to some extent. Some tribes accept the new ways of life with enthusiasm, but others resist them strongly.

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Sometimes neighboring tribes such as the Kikuyu and the Masai of eastern Africa react in opposite ways to Western influences. Many Kikuyu eagerly accept Christianity, European-type education and clothing, trade unions, and white bread. Although Kikuyu tribal law has forbidden eating fish, members of the tribe now do so. They saw Europeans eating fish, and decided it must be good. *Many Kikuyu in Nairobi* (where Vint's analyses were made), the capital of Kenya, are *prosperous businessmen, doctors, lawyers, and high-ranking politicians.* They drive their own cars and, *generally, live like well-to-do Europeans"* (emphasis mine).

The Vint series, as we have previously observed, consists of Nilotic Negroids, totaling 15 per cent of the whole. These people, as is well known, are literally the royalty of Black Africa. They are second to none in class and in political and economic power. They hold almost complete suzerainty over other African tribes along the White Nile. Since roughly two-thirds of Vint's series consists of Blacks, who in relation to their own strata are the socio-economic cream of Africa—indeed by world standards, they compare with the "best" from most anywhere—it is patent that this series is not underrepresentative of Blacks, quite the contrary, Vint's series is a very highly selective Black sample; thus attenuating possible racial differences. Even if the remaining one-third of the group were absolute dregs, and there is no evidence they were, the average status of the series is at least comparable to the White middle class. In addition, there were also Mullatoes in the series, a fact which, again, would tend to reduce possible brain differences.

Dr Tobias's suggestion that the series might be underrepresentative of Black status is untenable, absurd and untrue. Dr Tobias presents no evidence for rejecting the findings of F. W. Vint.

In summary, we can state without equivocation, that Dr Vint has carried out a reliable, competent and scholarly study of the Negroid cerebral cortex, a study in whose findings we may have confidence; a study of considerable merit and significance, which deserves the serious attention of all students of human neurology.

CONCLUDING THOUGHTS ON BRAINS AND RACE

It is felt, in this writer's opinion, that when a person continually contradicts himself (as we have seen Tobias has done), he is either trying to cover something up, or is afflicted with a marked impairment in judgment. In either case, Tobias's article surely marks one of the most disturbing pages in the history of anthropology or biology. Little more can be said on the main body of Tobias's paper. We have seen the contradictions,

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irrelevancies, misrepresentations, prevarications, the numerous undocumented assertions, the allegations, the periodic, indeed frequent or characteristic suspension of logic. We have seen that few of his conclusions are commensurate with the facts he presents and fails to present. We have seen that Tobias has little respect for facts, at least as far as this area of research is concerned. And finally, we have seen that his major allegation, that there are no demonstrated differences in the size and structure of the brains of Blacks and Whites, is pure mendacity.

It is most disheartening and unsettling to find, in this era of Watergate, still another coverup unfolding. We are reminded of the words of Thomas Jefferson: " To open the doors of truth, and to fortify the habit of testing everything by reason, are the most effectual manacles we can rivet on the hands of our successors to prevent their manacling the people with their own consent " (1804).

In closing the critique portion of this open paper, it is perhaps not inappropriate to cite the words of Dr Tobias himself, the closing words of his article, words which are tantamount to an unconscious, albeit spontaneous, open confession: " The exploding of the myth about brain-size and grey matter differences leads to the realization that, in science, the truth does not gain acceptance by mere repetition of a set of facts. Hypotheses are not confirmed by statement and re-statement of the hypotheses. Science requires rather the patient testing of the facts, the repeating of early studies by more modern, better controlled and better standardized methods, the constant re-examination and critical re-appraisal of premises and assumptions, the elasticity of mind which permits, *nay demands,* old hypotheses to be modified when new facts emerge which cannot be adequately explained by them, resistance to the tendency to develop a vested interest in a particular viewpoint, avoidance of ascribing motives to scientists of opposite viewpoint, in favour of the unbiassed examination of the evidence they may advance, the eschewing of premature hypotheses based on overtenuous evidence " (emphasis mine).

SUMMARY

(1) Tobias presents no evidence to invalidate the finding that Negroids have statistically and biologically significantly smaller brains than Whites.
(2) Tobias presents no evidence that body stature plays a significant rôle in observed brain weight differences between Blacks and Whites.
(3) Tobias presents no evidence that age plays any rôle in the

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observed differences in brain size between Blacks and Whites.

(4) Tobias presents no evidence that the nutritional circumstances of Negroids or Whites account for the relative measured differences in abstract intelligence or observed differences in brain size or structure.

(5) Tobias presents no evidence that non-nutritional environment accounts for observed differences in mental test levels, brain weight or brain structure, between Negroid and White.

(6) Tobias presents no evidence that the source of the brain sample can account for observed differences in brain size between Blacks and Whites.

(7) Tobias presents no evidence that occupational stratification accounts to any degree for observed brain weight differences between Negroids and Whites.

(8) Tobias presents no evidence that the cause of death accounts notably, if at all, for observed brain weight differences between Blacks and Whites.

(9) Tobias presents no evidence that the lapse of time after death and weighing the brain accounts, to any degree, for observed differences in brain weight between Blacks and Whites.

(10) Tobias presents no evidence that the treatment of the brain after death accounts to any degree for observed differences in brain weight and structure between Blacks and Whites.

(11) Tobias presents no evidence that a large brain, other factors being equal, as a rule, is not beneficial for survival or intellect, or that a small brain, other factors being equal, as a rule, is beneficial for survival or intellect.

(12) Tobias presents no evidence of a general evolutionary trend towards smaller brain size.

(13) Tobias presents no evidence that there are not differences in the depth of various areas of cerebral cortex between Blacks and Whites.

(14) Tobias presents evidence bearing on " items " 1 to 13 which point to, *nay demand,* the exact opposite conclusions he reached.

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PROFESSOR DOTT. MARIO CAPPIERI

We have pleasure in being able to announce that our colleague Professor Dott. Mario Cappieri of Rome has received the Thomas Nelson Annandale Memorial Gold Medal for his studies on Asian populations, from the well-known institution, the Asiatic Society of Bengal. We wish to add our tribute to his deserved excellence.

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Sir Grafton Elliot Smith on "the Negro Race"

By DONALD A. SWAN

Sir Grafton Elliot Smith (1871-1937) was born in Australia and was for many years Professor of Anatomy at the University of London, England. Described by the noted American anthropologist, Dr E. A. Hotton (1946),[1] as ". . . one of the greatest authorities on the evolution of the brain," Dr Elliot Smith conducted original studies on the anthropology of the brain of the living races of man.[2] He also contributed to our knowledge of the brains of fossil men by means of comparative study of their endocranial casts.[3] Professor Elliot Smith's work on human evolution and the evolution of the brain was best summarized in his volume *Essays on the Evolution of Man* (1927). His work in these areas is still highly regarded by students of the brain, such as Gerhardt von Bonin (1950).[4]

In addition to his writings and research in the field of physical anthropology, Sir Grafton Elliot Smith wrote several books dealing with various aspects of cultural anthropology or ethnology. These volumes included: *The Migrations of Early Cultures* (1915), *The Ancient Egyptians and the Origin of Civilization* (1923), and *The Diffusion of Culture* (1933). As an ethnologist, Dr Elliot Smith is classified as a member of the "British Diffusionist School." In his writings on ethnology, he contended that civilization originated in Ancient Egypt and that the important elements of culture were later diffused to other peoples and civilizations.

Contemporary students of prehistory, however, would not accord such a priority to the Ancient Egyptians. Clark (1961), for example, regards cereal-farming and stock-raising as originating

[1] Ernest A. Hooton, *Up From the Ape*, Macmillan, New York, 1946, p. 141.

[2] Grafton Elliot Smith, "The 'limbus postorbitalis' in the Egyptian Brain," *Anatomische Anzeiger*, Vol. 24, 1903, p. 139; *Studies in the Morphology of the Human Brain, with Special Reference to that of the Egyptians. No. 1: The Occipital Region*, Records, Egyptian Government School of Medicine, 1904.

[3] Grafton Elliot Smith, "Endocranial Cast Obtained from the Rhodesian Skull," *Rhodesian Man and Associated Remains*, British Museum of Natural History, London, 1928, pp. 52-58; J. L. Shellshear and Grafton Elliot Smith, "A Comparative Study of the Endocranial Cast of Sinanthropus," *Philosophical Transactions of the Royal Society of London, Series B*, Vol. 223, 1934, pp. 469-487.

[4] Gerhardt von Bonin, *Essay on the Cerebral Cortex*, C. C. Thomas, Springfield, Illinois, 1950, p. 9.

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in western Asia and being diffused later to Egypt.[5] On the other hand, Dr Elliot Smith's view that "the . . . debt of Africa to Egypt is beyond queston"[6] is fully in accord with contemporary archaeological opinion.

Sir Grafton firmly believed that there exists "an intimate relationship between Anatomy and Human History."[7] In 1934 Dr Elliot Smith published a 500-page volume entitled *Human History*, which provided a synthesis of "Biology and the Humanities to interpret human thought and action."[8] The book contains 15 chapters ranging from "The Wanderings of Primitive Men" to "The World-Wide Influence of Greece." In his chapter on "The Races of Mankind," Professor Elliot Smith outlined the biological and cultural traits of the primary races of man. The section on "The Negro Race," which is discussed in the following part of this article, contains as concise and as accurate a description of the traits of the Negro as is to be found in the anthropological literature. In view of contemporary journalistic trends to falsify racial history,[9] it is worthwhile to review the writings of a scientist who was one of the greatest authorities of this century in the fields of human anatomy and biology.

Professor Elliot Smith began his chapter on "The Negro

[5] Graham Clark, *World Prehistory: An Outline*, Cambridge University Press, New York, 1961, pp. 99-128.

[6] Grafton Elliot Smith, "The Diffusion of Culture," *Culture: The Diffusion Controversy*, W. W. Norton, New York, 1927, p. 20.

[7] Grafton Elliot Smith, *Human History*, Jonathan Cape, London, 1934, p. 14.

[8] *Ibid.*

[9] Robert Gayre, "Negrophile Falsification of Racial History," THE MANKIND QUARTERLY, Vol. VII, No. 3, January-March 1967, pp. 131-143; Hugh Trevor-Roper, *The Rise of Christian Europe*, Thames and Hudson, London, 1965, pp. 9-11:

"It is fashionable to speak today as if European history were devalued: as if historians, in the past, have paid too much attention to it; and as if, nowadays we should pay less. Undergraduates, seduced, as always, by the changing breadth of journalistic fashion, demand that they should be taught the history of black Africa. Perhaps, in the future, there will be some African history to teach. But at present there is none, or very little: there is only the history of the Europeans in Africa. The rest is largely darkness, like the history of pre-European, pre-Columbian America. And darkness is not a subject for history."

". . . The new rulers of the world, whoever they may be, will inherit a position that has been built up by Europe, and by Europe alone. It is European techniques, European examples, European ideas which have shaken the non-European world out of its past—out of barbarism in Africa, out of a far older, slower, more majestic civilization in Asia; and the history of the world, for the last five centuries, in so far as it has significance, has been European history. . . ."

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Race " with a brief review of the physical and anatomical traits of the race. He noted that the " Negro is a very primitive member of the human famly, and yet in some respects is highly specialized. The average size of the brain is much smaller than that of the peoples of Europe and Asia. Moreover, anatomical peculiarities suggesting affinities with the apes are commoner than they are in most other peoples."

With respect to hair form, Dr Elliot Smith found the Negro to be highly specialized. Viewed in cross-section the hair of a European is seen to be elliptical. As a result of this flattening European hair tends to become either wavy or curly. " In the Negro the flattening is carried much further, so that the hair becomes almost ribbon-shaped." Consequently, Negro hair " tends to become rolled up in tightly curled masses, commonly called ' peppercorns.' " According to Elliot Smith, " no other race has hair that is so flat or tightly curled."

In contrast to the hair of the Negro, the hair of the Mongol " is almost circular in section, and is considerably coarser than that of any other race; it is quite straight, showing no tendency whatever to become wavy." Thus, as regards hair form, the Negro and the Mongol " are highly specialized in different directions." The distinctive hair form of the Negro also serves to distinguish this race from the Australoid race, which resembles the Negro in skin-colour and form of the nose.

The Negro race also differs from the Australian aboriginals in respect to the brow ridges over the eyes. In the Australian " there is a tendency for the size of the eyebrow ridges to become exaggerated. . . . In the Negro the eyebrow ridges are singularly defective. They are, in fact, even less noticeable than in the European."

Sir Grafton also found the bones of the Negroes to be " peculiarly specialized. They are more slender and denser than those of other races. Less robust in build, the limb bones depend for their strength upon their dense, ivory-like texture."

Professor Elliot Smith concluded that the " Negro and the Australian are more primitive than any other living peoples." However, the two races differ in many physical features. " The Negro is characterized by a singular lack of hair; the pure Negro very rarely has more than a few tufts of hair for a beard. The Australian sometimes has, not only a thick beard, but often a great development of hair on the body."

In skin colour both the Negro and Australoid races " have retained the dark skin pigment which probably was the heritage of all human beings." In the northern Caucasoid and Mongoloid races, " the development of the colouring matter in the skin is hindered, so it appears as though it had passed through a sort of

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bleaching process." Thus, the yellowish-brown colour of races "like the Mediterranean, the Bushman, and the Mongol is due to a partial bleaching of the original black skin common to the African anthropoid apes and to the earliest members of the human family."

The shape of the Negro nose is another distinctive racial feature. Resembling that of the Australian aborigine, the nose of the Negro "is very flat, and the wings of the nostrils are broadly expanded." This extreme nasal flatness is also seen in certain branches of the Yellow or Mongol race, "but these Asiatics lack the broadly outsplayed nostrils of the African and Australian races." Dr Elliot Smith believed that "the flattening of the nose in Australians, Negroes, and Mongols probably represents the retention of a primitive trait, which is still manifest in the human foetus of every race."

Another characteristic of the Negro race "is the full, fleshy lips." In the case of this trait, however, Dr Elliot Smith believed that "it is a sign of high specialization distinctive of the Negro," rather than a primitive feature. In comparison, the lips of the anthropoid apes are thin, not fleshy.

Sir Grafton also noted that "the muscles of the face in the Negro and Australian races are not so highly differentiated as those of Europeans, and are less apt to express subtle shades of emotion than those of the other races of mankind. The Negro's facial suppleness is less varied and less subtle, and his robuster manifestations of mirth and sorrow more unrestrained."

The Negro race shows the most extreme variations in stature. "The tallest and shortest people in the world both belong to the Negro race." The two extremes, who differ in mean stature by nearly two feet, live in close proximity to one another. The extremely tall group, the Nilotic Negroes of the southern Sudan, live near the head-waters of the Nile River. They include tribes such as the Shilluks and Dinkas. The Pygmies inhabit Equatorial Africa not far to the south. In the view of Dr Elliot Smith, "both these groups are Negroes, with the typical hair, skin-colour, full lips, and other distinctive morphological characters." Most other African Negro groups are intermediate in stature, averaging about five feet six inches in height.

Anthropologists have long been interested in the taxonomic relationship of the dark-skinned peoples—both normal-statured and dwarfed—of Southeast Asia to those of Africa. For Carleton Coon (1962), the dark-skinned peoples of Southeast Asia are Australoid in origin. They are the end-product of a separate evolutionary line derived from *Pithecanthropus erectus.* In Dr Coon's view, the living Australoid subspecies is divided into three races: (1) the Australoid proper, full-sized with straight or wavy

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hair; (2) the Tasmanian and Papuo-Melanesian, full-sized with negroid hair; and the Negritoes, pygmy-sized, with negroid hair.[10]

Sir Grafton Elliot Smith, however, regarded the peoples of New Guinea and Melanesia as Negroes, sharing a common origin with the Pygmies and Negroes of Africa. Thus, he believed that "the Negro population of the world was in ancient times broken up into two main groups, African and Melanesian, widely separated in the geographical sense." In terms of relative population numbers, "the vast bulk of the Negro Race is to be found in Africa, where it occupies the tropical belt as far north as the Sahara, and spreads up the west coast almost as far as Morocco."

The other great mass of the Negro race was viewed as inhabiting New Guinea and spreading "right through Melanesia, the Bismarck Archipelago, the Solomon Islands, New Caledonia, and Fiji, and even to Easter Island, far out in Eastern Polynesia, and the American coast." The Melanesian Negroes were described as presenting the general characteristics of the African Negro, "but in a form suggesting more or less admixture with other races." As in Africa, dwarfed or short-statured peoples—termed Negritoes—are also found among the Melanesians.

In the intervening area between the African and Oceanic Negroids, only small numbers of Negroid peoples are found. These include the inhabitants of the Andaman Islands—lying to the south of Bengal and Burma. For Sir Grafton the question arose as to the location of the original home of the Negro: "Did the Negro acquire his distinctive traits in Africa and spread to the Far East, or in the Far East and spread west? Or was his original home in some region between East Africa and Papua?"

Professor Elliot Smith noted that ". . . Africa contains the vast majority of Negroes at the present time, and that in Africa the Negro is found with relatively less evidence of extreme admixture than elsewhere." The distinctive features of the Negro Race "are revealed in a much more obtrusive and constant form in Africa." These facts suggested that "Africa was their original home." Then, Negroes supposedly reached Papua and Melanesia in early times, "after wandering along the southern littoral of Asia."

In his review of *The Origin of Races* (1962), Carleton Coon speculated that the earliest population of India "was probably Australoid in the wide sense." He observed that in India today "a minority of the Dravidian speakers, composed mostly of tribal peoples outside the caste system, are Australoid, and a very few are Negroid." Both groups were believed to have adopted

[10] Carleton S. Coon, *The Origin of Races*, A. Knopf, New York, 1962, pp. 425-427.

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Dravidian languages from their culturally more advanced neighbors.[11]

For Sir Grafton Elliot Smith the one great factor in the population of India was "the presence of members of the Australian Race called Pre-Dravidian." However, he also noted traces "of intermingling with the Negro Race, to which part of the darkness of skin-colour so widely prevalent in India may be attributed." And Dr Elliot Smith believed that there were "clear signs of Negro mixture in Southern Persia, where the people show a marked contrast to those in Northern Persia."

Sir Grafton began his review of the various branches of the Negro race and their geographical distribution with the short-statured or dwarfed peoples of Africa. The really negroid Pygmies are divided into two groups—one in Equatorial Africa and the other in the Congo. They are the shortest people in the world and inhabit the tropical rain forests. According to Dr Elliot Smith, the Pygmies "are extremely primitive, being quite devoid of any kind of culture. They are skilled in hunting, and they have no other arts or crafts; they wear no clothes; they build no houses; their needs are few, and their activities extremely limited in scope."

The taxonomic relationship of the short-statured, lighter-skinned Bushmen and Hottentot peoples of southern Africa to the darker-skinned Negroid peoples of the continent has also interested many anthropologists. For Carleton Coon (1962), "the living Bushmen, the living Hottentots and that branch of the Hottentots known as the Korana, a few beachcombing remnants of an earlier coastal Bushman population known as Strandloopers, and certain relict populations in Tanganyika and possibly farther north" comprise one of the five subspecies of modern man. They are collectively termed the Capoids. In Professor Coon's view the living Bushmen and Hottentots form a separate evolutionary line "descended from the Ternefine-Tangier line of North Africa." Furthermore, he contends that their ancestors were driven out of North Africa "by an invasion of Caucasoids toward the end of the Pleistocene and during the early post-Pleistocene period; and that they did not begin to undergo a reduction in size until after arriving in their historic homeland, South Africa, Southwest Africa, Bechuanaland, and the Rhodesias."[12]

Professor Elliot Smith, however, regarded the Bushmen and Hottentots as part of the Negro race. He noted that "their skin is not black but yellow in colour, rather like that of a typical

[11] Carleton S. Coon, *op. cit.*, p. 422.

[12] Carleton S. Coon, *op. cit.*, pp. 636-639.

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Mongol. . . . " Nevertheless, he concluded that apart from skin-colour, the Bushmen and Hottentots are " truly Negro in type."

The hair of the Bushmen and Hottentots was described as ultra-Negroid. The " peppercorn " type of hair is seen in its most extreme form in the case of the Bushmen. According to Sir Grafton, " the head looks as if it had been shaved and peppercorns stuck on so as to leave spaces in between them." The nose of the Bushman " is even flatter than that of the ordinary Negro." Consequently, Dr Elliot Smith concluded that the Bushman could be described as a diminutive Negro, " with all the characteristic features of the race carried to the extreme, with the exceptional distinction that the skin is bleached to yellow."

A distinctive trait of the Capoid race is " steatopygy." According to Dr Elliot Smith, " In the women of the Bushmen and Hottentot peoples, enormous masses of fat develop in the region of the buttocks and hips, giving the form of the body a monstrous and often grotesque appearance. It differs from the condition that is frequently found in women of all races, in whom an unusual obesity produces a distortion of the whole body." True steatopygy is not due to obesity, " but to the localized deposition of large masses of fat in one region of the body." Furthermore, steatopygy is normal throughout the Capoid race, rather than an individual peculiarity.

According to Carleton Coon (1962), a number of post-glacial fossil crania found in South Africa are " racially non-Capoid." These include the Cape Flats and Border Cave skulls. Dr Coon reports that South African anthropologists classify these crania as " Australoid." If such an element existed in South Africa in the past, it could be the source for the appearance of " Australoid " traits in some living African peoples. These include the Korannas of the Orange River region of South Africa, who are regarded by Carleton Coon as " a kind of Hottentot."[13]

Professor Elliot Smith described the Korannas as having a darker skin and generally showing more signs of Negro affinity than the Bushmen or Hottentots. He believed the Korannas worthy of notice, " because, unlike other Negro peoples, they show a tendency to develop prominent eyebrow ridges." Thus, they present " resemblances to the Australian Race." Sir Grafton suggested that the Korannas may have " intermingled with some more primitive race which existed in former times in Africa."

In earlier times the Bushmen and Hottentots extended further to the north in Africa than their present homeland. As noted by Professor Elliot Smith, " the Bushmen have been gradually pushed by the Bantu Negroes towards the south, and particularly into the Kalahari Desert, where they exist today." Some writers

[13] Carleton S. Coon, *op. cit.*, pp. 630-631.

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have speculated that in prehistoric times the Bushmen even inhabited southern Europe, North Africa and Egypt. These speculations were based upon finds of Bushmen-like rock paintings in the Sahara, statuettes depicting corpulent women, and alleged Bushmen-like traits in some of the oldest ancient Egyptian skulls.

Professor Elliot Smith rejected these theories. He contended that " the form of these statuettes had nothing to do with race, but was associted with the ideas about the power of a female amulet to give life and protect those who used the image." Sir Grafton also rejected the claim of Marcellin Boule that the skeltons in the Grimaldi caves of southern France were negroid and once belonged to Bushmen-like people. He concluded that " the skeletons from the Grottes des Enfants did not belong to members of the Negro Race, but simply showed the retention of primitive features more nearly akin to those of the aboriginal Australians."

Professor Elliot Smith divided the Negro peoples of Africa into two major groups—the Sudanese Negroes and the Bantu Negroes. The Sudanese Negroes inhabit Equatorial Africa " from the head waters of the Nile to Nigeria and the Gold Coast." The Bantu Negroes occupy the region from the Great Lakes southward. Sir Grafton distinguished between the two groups of Negroes largely, but not wholly, on the basis of language. He also observed physical differences between the Sudanese and Bantu Negroes which are " partly due to the contrast in the process of intermingling with neighbouring races that has taken place in the two cases."

In his survey of *The Living Races of Man* (1965), Carleton Coon contended that the living African Negroes are " a product of mixture between invading Caucasoids and Pygmies who lived on the edges of the forest, which at the end of the Pleistocene extended farther north and east than it does now. To this combination may have been added remnant Capoid genes acquired in the Sahara and East Africa." He also suggested that " such a mixture has been going on for at least 15,000 years. . . ."[14]

Professor Elliot Smith also pointed out that " for thousands of years there has been a steady traffic to and fro across Equatorial Africa of members of the Mediterranean Race (Hamites and Arabs). The result of these movements has been the introduction into the Negro population of considerable Hamitic and Semitic elements, as well as a variety of cultural influences.

[14] Carleton S. Coon, *The Living Races of Man*, A. Knopf, New York, 1965, pp. 122-125.

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Every kind of intermingling has taken place between the original groups of Negro, Hamitic, and Semitic peoples."

Dr Elliot Smith believed that the Mediterranean branch of the White race "originally extended right up the Nile and the coast of East Africa to Somaliland and Equatorial Africa." At the head of the Blue Nile are to be found war-like Hamitic peoples, who are described as retaining "their Mediterranean characteristics with relatively slight admixture." Sir Grafton contended that "in early ages this war-like group intermingled with the neighbouring Negroes, to whom they gave leaders and taught them the art of war." As a consequence, two influences are seen to have been at work in Africa—"intermingling in the Southern Bantu region with the war-like Hamitic group, and in the Northern Sudanese area the more peaceful penetration by peoples from the east and north in search of raw materials."

Professor Elliot Smith regarded the Far Eastern group of Negroes in Papua and Melanesia as "even more mixed than the African group." In particular, the Melanesians were viewed as becoming mixed with people of Australoid and Mediterranean affinities during their easterly wanderings. Sir Grafton also believed a negroid element to be present in the South Pacific—from New Zealand to Easter Island. The presence of pygmy Negritoes in the Andaman Islands and in various parts of the Malay Archipelago as far east as the Philippines and New Guinea was also regarded as evidence of earlier easterly migration by Negroid peoples.

The relation of the Negro and other primitive peoples of sub-Saharan Africa to the more advanced peoples and cultures of Europe and the Mediterranean region may best be described in terms of retarded evolution, psychological deficit, and cultural lag. Throughout the long Palaeolithic period the tool-traditions of Africa lagged substantially behind those of Europe and South-west Asia. The European Upper Palaeolithic cultures (B.C. 30,000 to 10,000) with their elaborate and complex blade tools (especially the Solutrean culture of France), composite tools, extensive use of bone, and the first appearance of art (cave paintings and sculptured figurines), symbol and notation have no counterpart in sub-Saharan Africa.

In some parts of Africa primitive stone-tool cultures persisted until historic times and even into the present century. The use of metal comes late to sub-Saharan Africa and is introduced from outside the continent. According to Professor Coon, there was no Bronze Age in Africa, except for Egypt and a few Mediterranean coastal sites. Iron is introduced to Egypt from Asia Minor early in the first millennium B.C. and then slowly diffused southward.[15]

[15] Carleton S. Coon, *Living Races of Man*, pp. 88-90.

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Carleton Coon also notes that " in post-glacial times the archaeological history of food-gathering cultures in Africa consists . . . of a gradual drifting southward of industries originating in the north." When food-producing cultures eventually emerge in sub-Saharan Africa, virtually all of the domesticated plants and animals are introduced from outside the continent. The domesticated plants of contemporary tropical Africa were brought from Southeast Asia by Indonesians in early historic times and from the Americas by Europeans in the colonial period. The former introduced into Africa taro, yams, banana plants, sugar cane, Asiatic rice, and coconuts. The latter introduced maize, manioc, sweet potatoes, peanuts, pumpkins, squash, cacao, and tobacco.[16]

Sir Grafton Elliot also reviewed the cultural achievements of the Negro race. He concluded that:

> . . . Neither in Africa nor in Melanesia can any significant element of culture be attributed to Negro invention. Many of them, like the African Pygmies and the Indonesian and Papuan Pygmies, seem to be almost wholly devoid of any culture. As regards the other Negroes in Africa, who for more than 40 centuries have been in contact with the pioneers of civilization, the few arts and crafts that are scattered sporadically through the vast continent are all alien in origin. No more striking testimony is needed to emphasize the lack of enterprise and initiative in the Negro Race.

Although written more than 40 years ago, the words of Dr Elliot Smith should be read today by everyone who is interested in the continent of Africa and its future possibilities of development. For in the final analysis, economic and social development depend upon the biological abilities of the inhabitants of the area. Assessment of these abilities requires accurate knowlodge of the biological and cultural traits of the various races and peoples—which in the case of the Negro race has been well-provided for us by Sir Grafton Elliot Smith.

[16] Carleton S. Coon, *Living Races of Man*, pp. 89-91.

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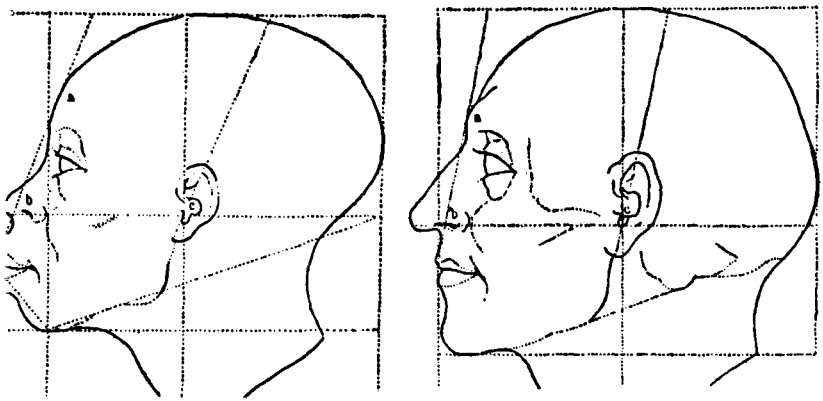

FIGURE 1—Racial differences in facial prognathism — Camper Angle (Canella, 1943). Left: Prognathous African Negro; right, Orthognathous European White.

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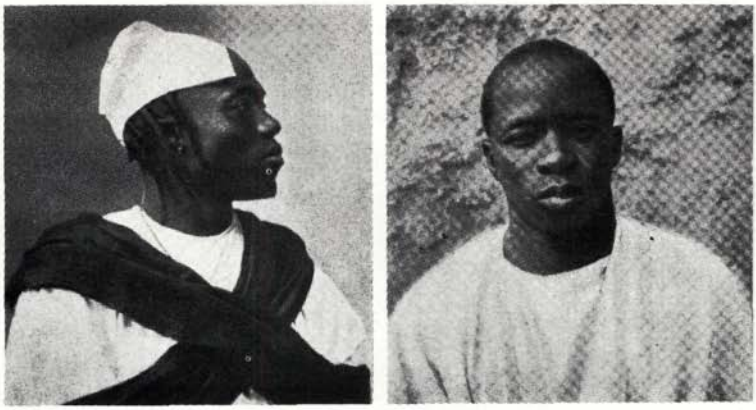

FIGURE 3—Sudanese Negroes. Bambara man (left) and Soninke man from West Africa (Biasutti, 1959).

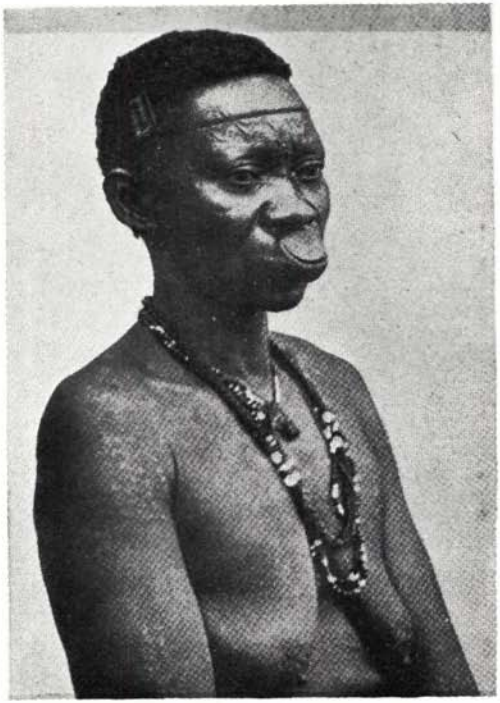

FIGURE 4—Bantu Negro. Yao woman from East Central Africa (Biasutti, 1959).

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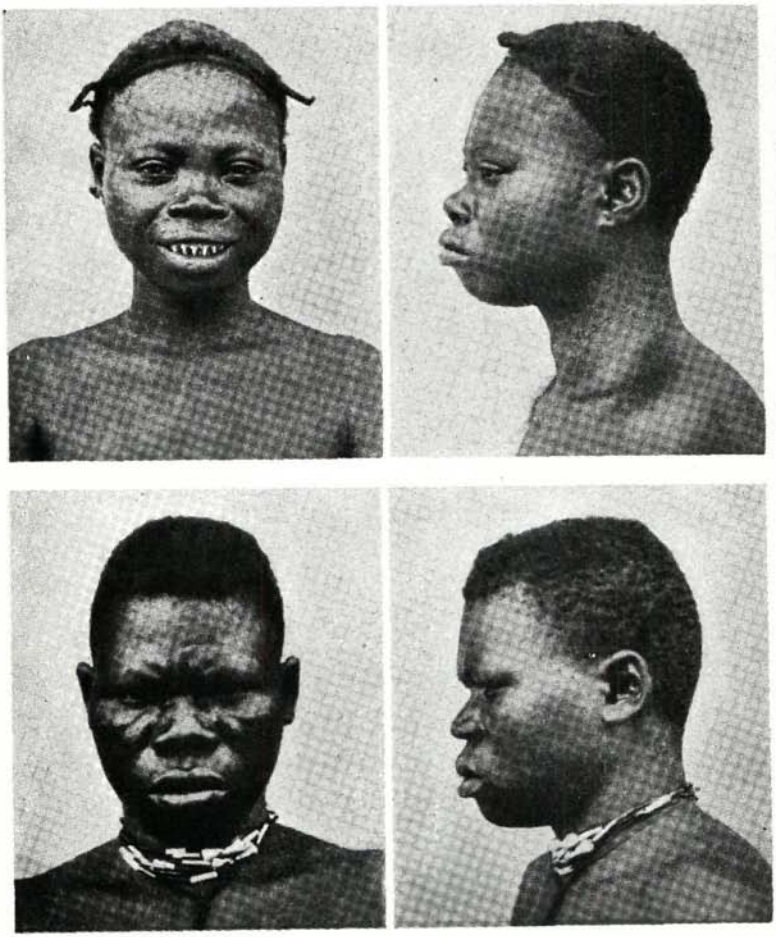

FIGURE 5—Forest Negroes. D'zem man (top) and Kongo man (bottom) from Central Africa (Biasutti, 1959).

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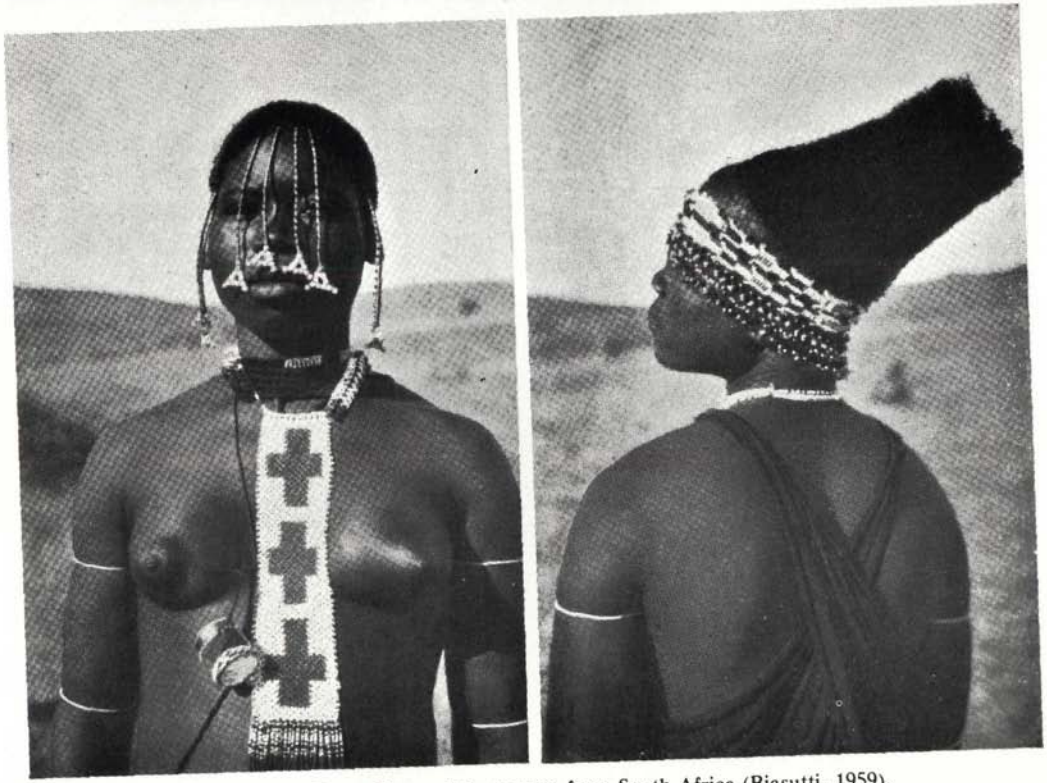

FIGURE 6—Bantu Negro. Zulu woman from South Africa (Biasutti, 1959).

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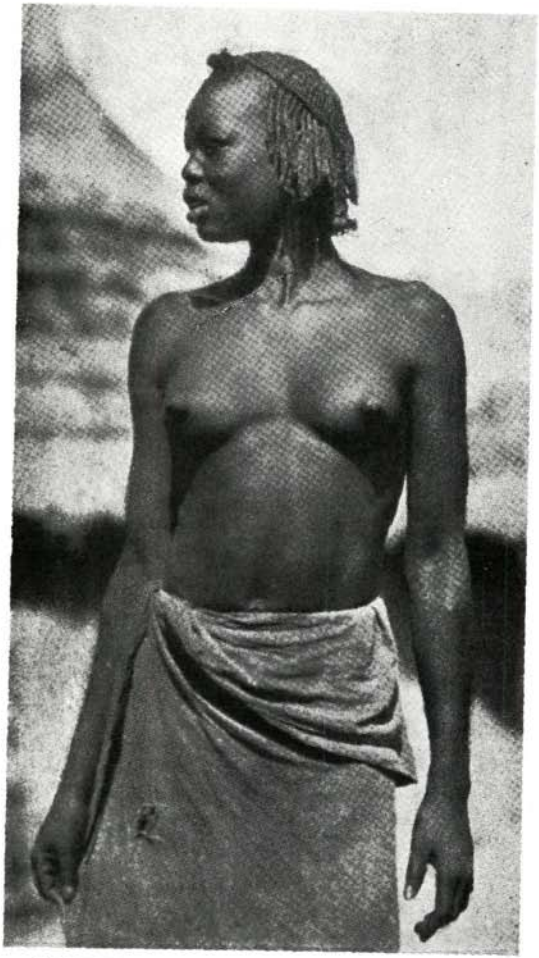

FIGURE 7—Nilotic Negro. Nuba woman from the southern Sudan (Biasutti, 1959).

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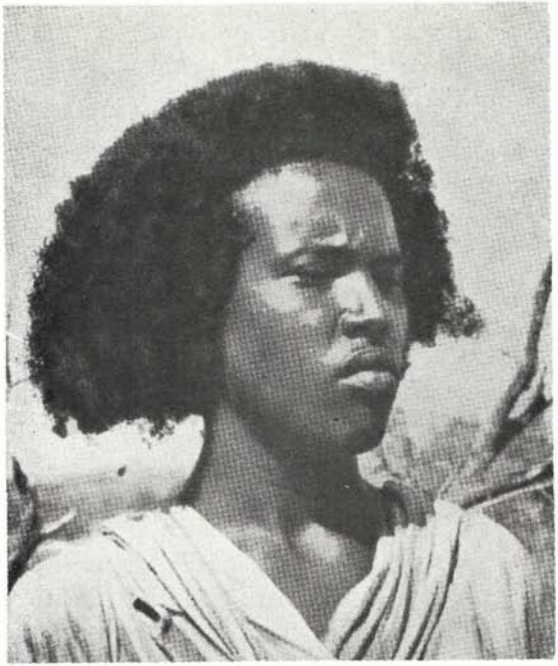

FIGURE 8—Ethiopid Negro. Somalo Gherra man from Ethiopia (Biasutti, 1959).

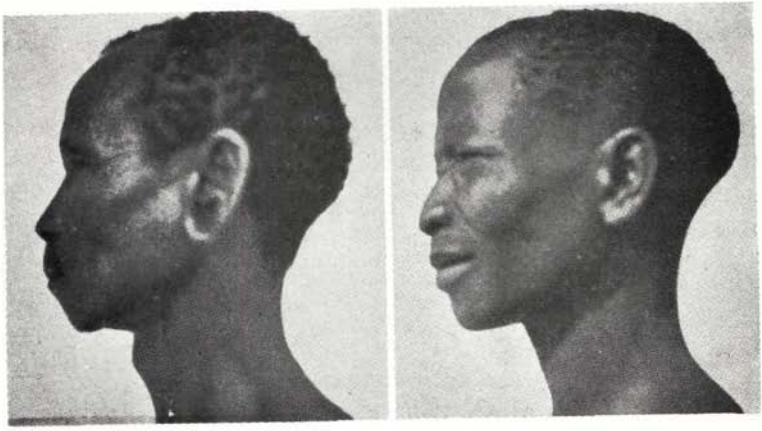

FIGURE 9—Capoid relict population. Two Sandawe men from Tanganyika (Biasutti, 1959).

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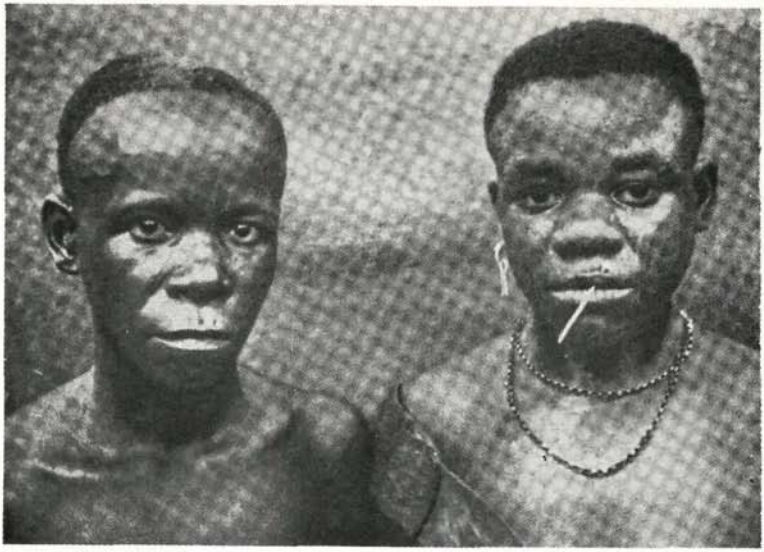

FIGURE 10—African Pygmies. Two adult Pygmy men from the Central African forest region (Biasutti, 1959).

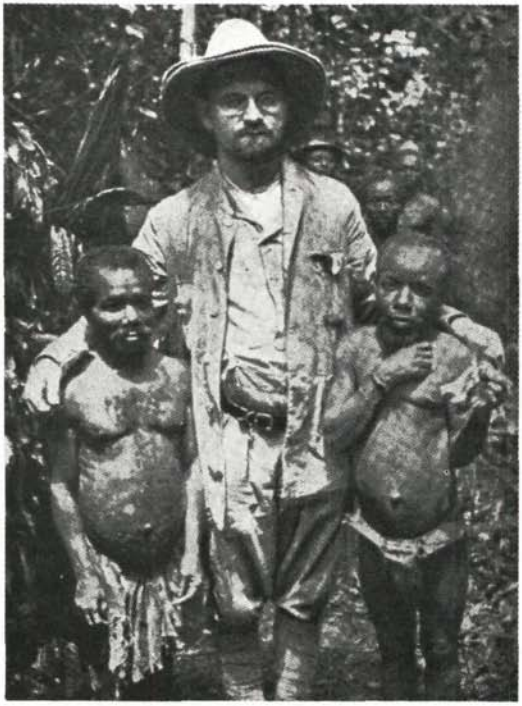

FIGURE 11—African Pygmies. Two Basua Pygmies with the anthropologist Dr Paul Schebesta (Biasutti, 1959).

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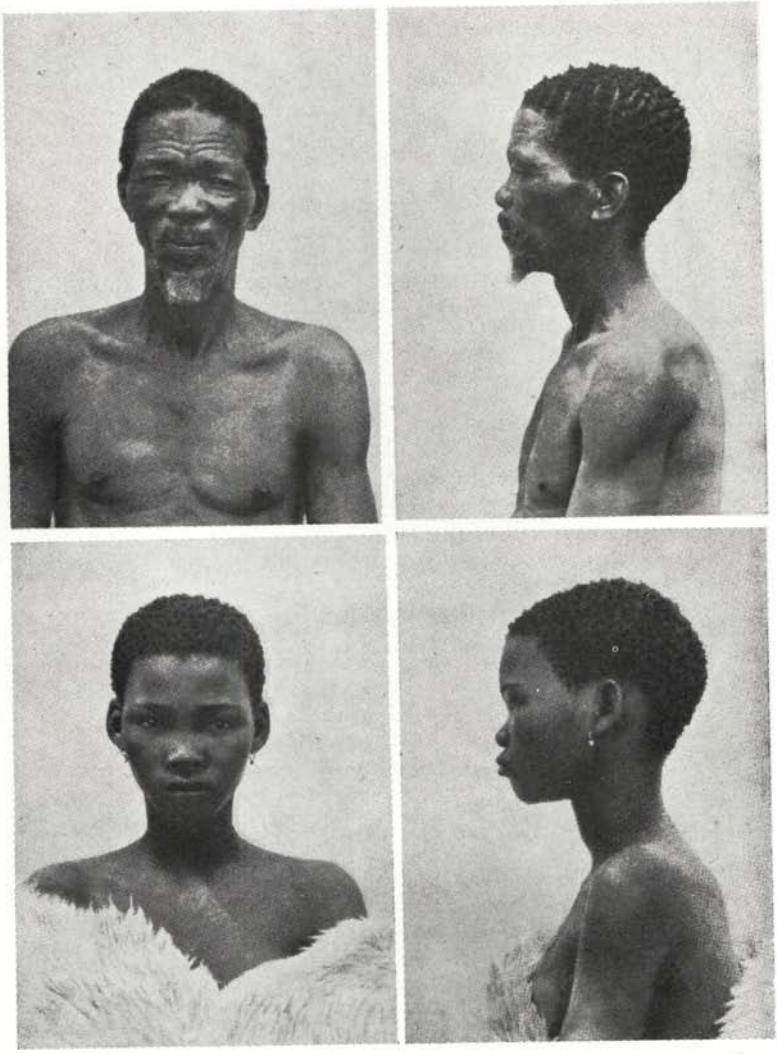

FIGURE 12—South African Hottentots. Topnaar Hottentot man (top) and Topnaar Hottentot woman (bottom) from Southwest Africa (Biasutti, 1959).

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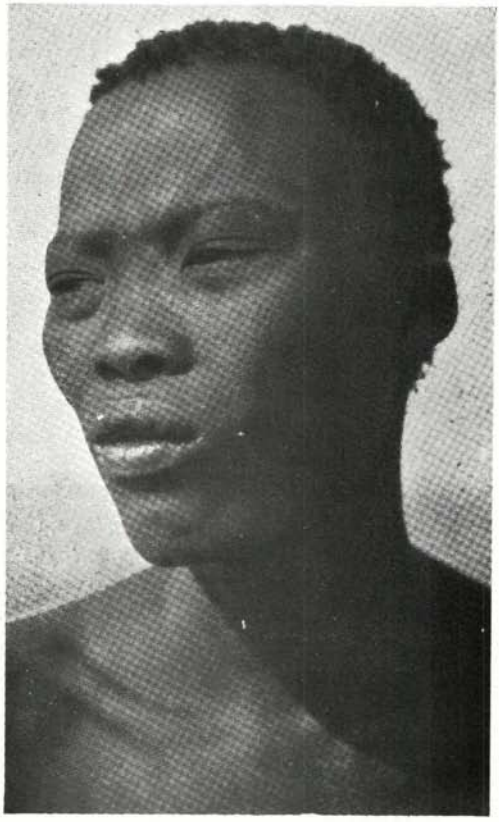

FIGURE 13—South African Bushman. Kung Bushman from Kalahari Desert region in South Africa (Biasutti, 1959).

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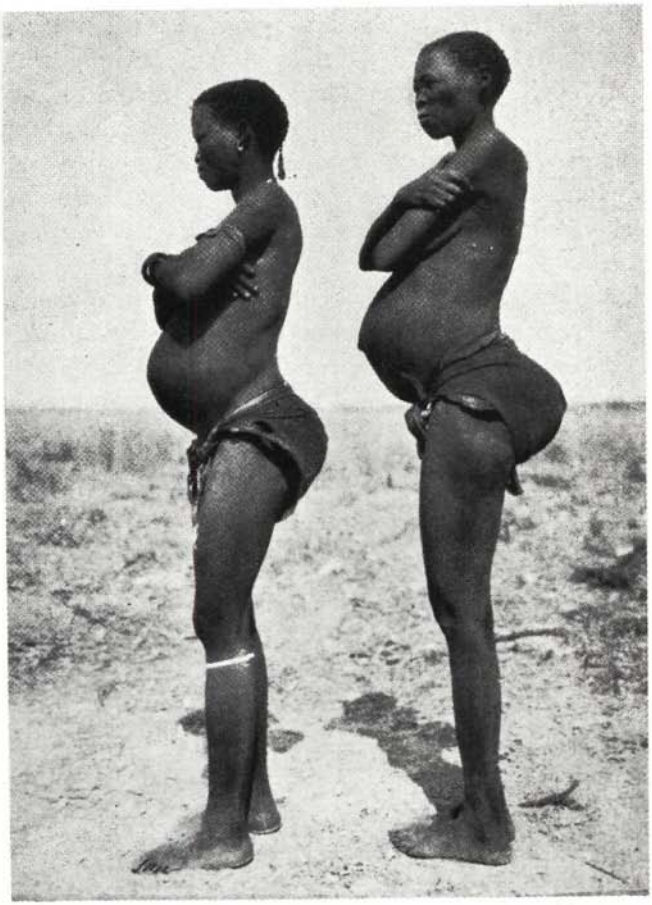

FIGURE 14—South African Bushmen. Two Kung Bushmen women from the Kalahari Desert region in South Africa (Biasutti, 1959).

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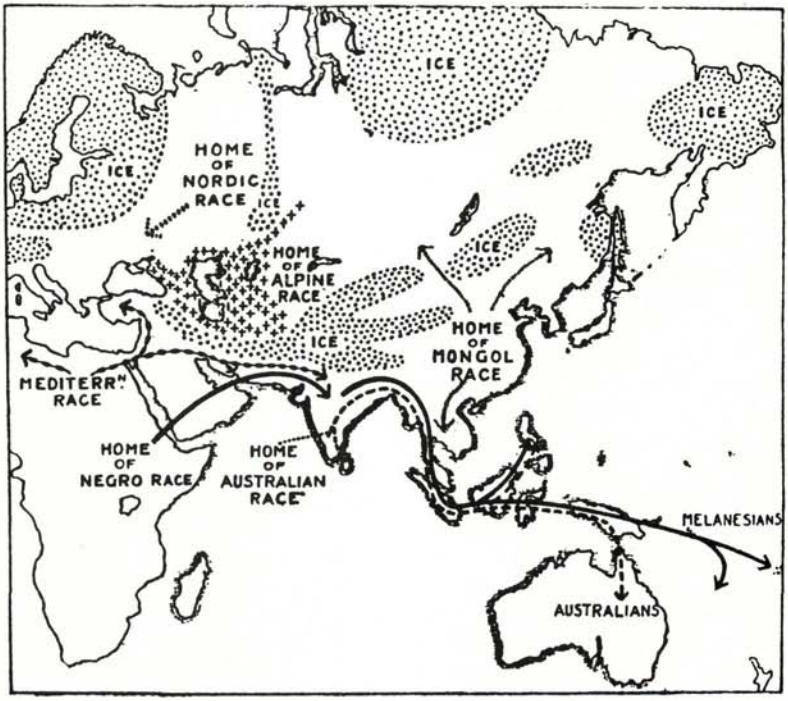

FIGURE 15—Early migrations of races, as postulated by Sir Grafton Elliot Smith (Elliot Smith, 1934).

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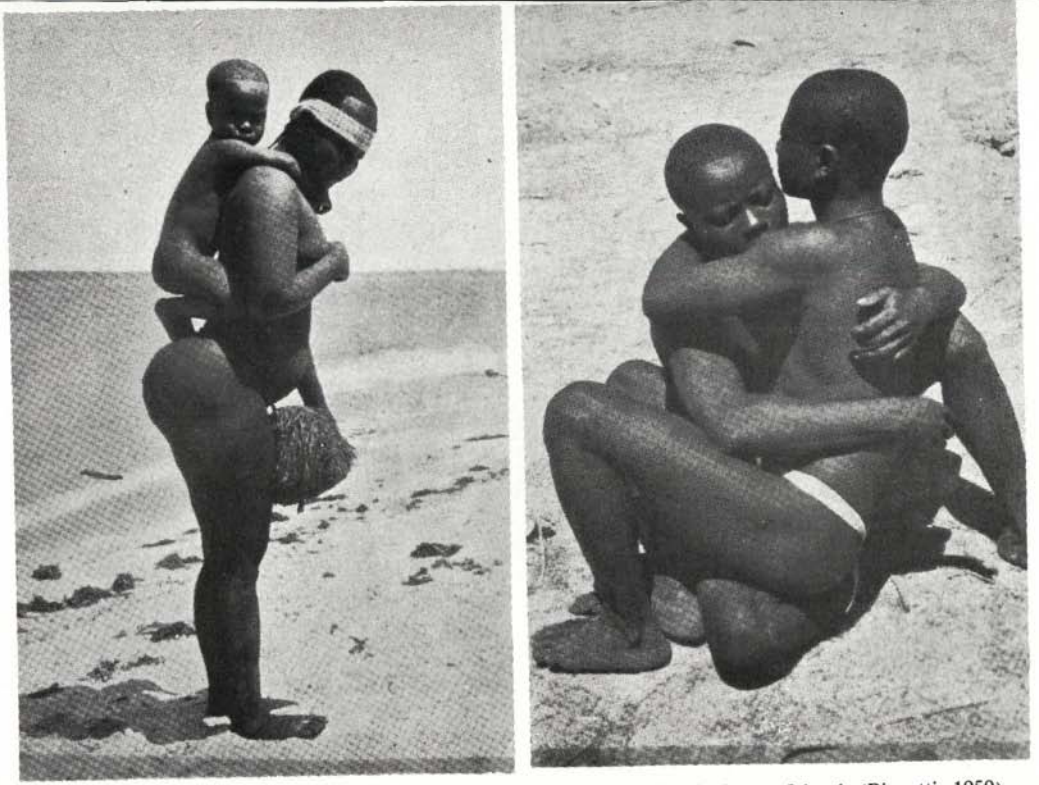

FIGURE 16—Southeast Asian Negritoes. Onghi Negritoes from the Andaman Islands (Biasutti, 1959).

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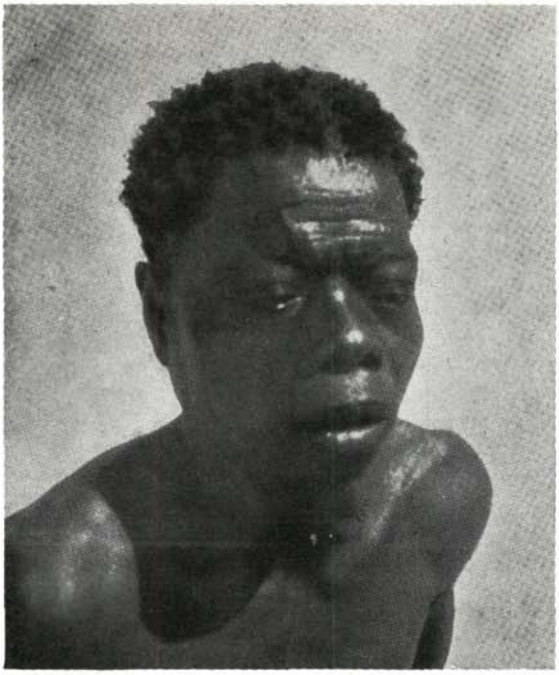

FIGURE 17—Southeast Asian Negrito. Onghe man from the Andaman Islands (Biasutti, 1959).

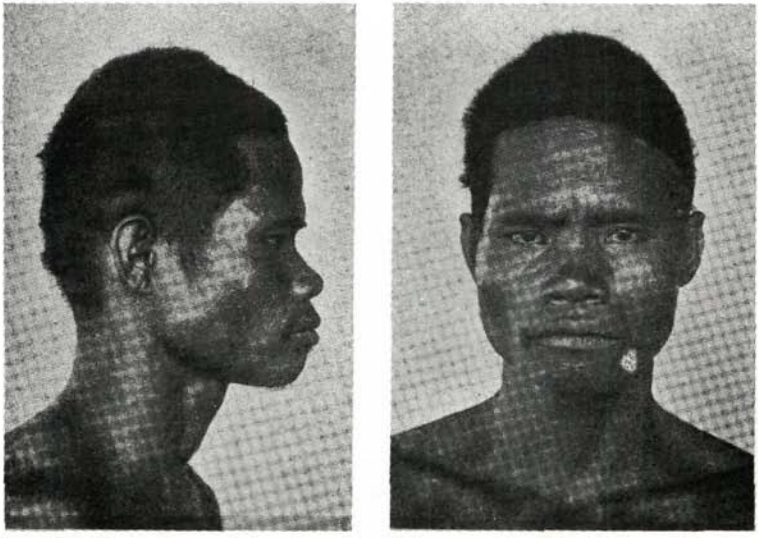

FIGURE 18—Southeast Asian Negroids. Tokea man from the Celebes Islands in Indonesia (Biasutti, 1959).

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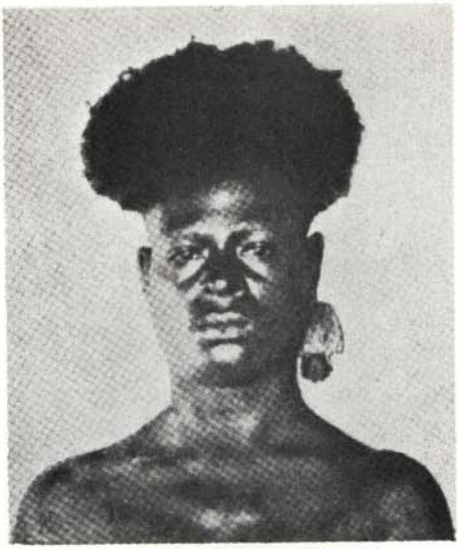

FIGURE 19—Melanesian Negroid. Yabim man from Finch Harbor, New Guinea (Biasutti, 1959).

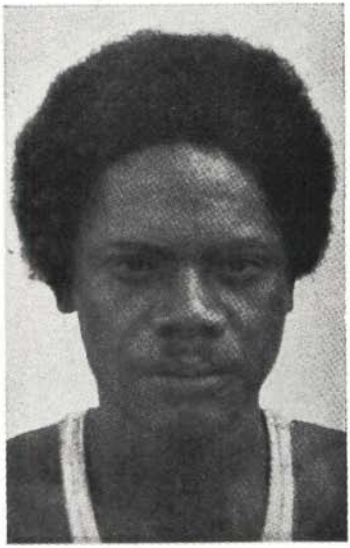

FIGURE 20—Southeast Asian Negrito. Manide Aeta man from Malaya (Biasutti, 1959).

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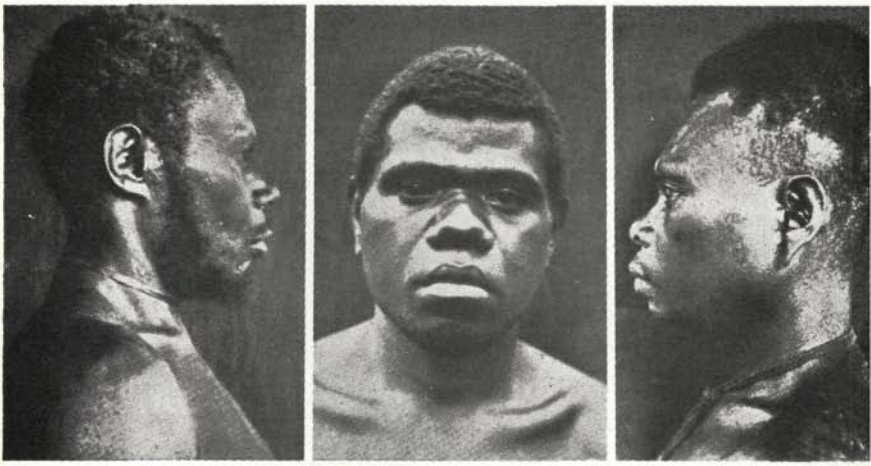

FIGURE 21—Melanesian Negroids. Melanesian man (left) from New Britain; Melanesian man from New Ireland; Melanesian man (right) from New Britain (Biasutti, 1959).

FIGURE 22—Southeast Asian Negrito. Semang man from Malaya (Biasutti, 1959).

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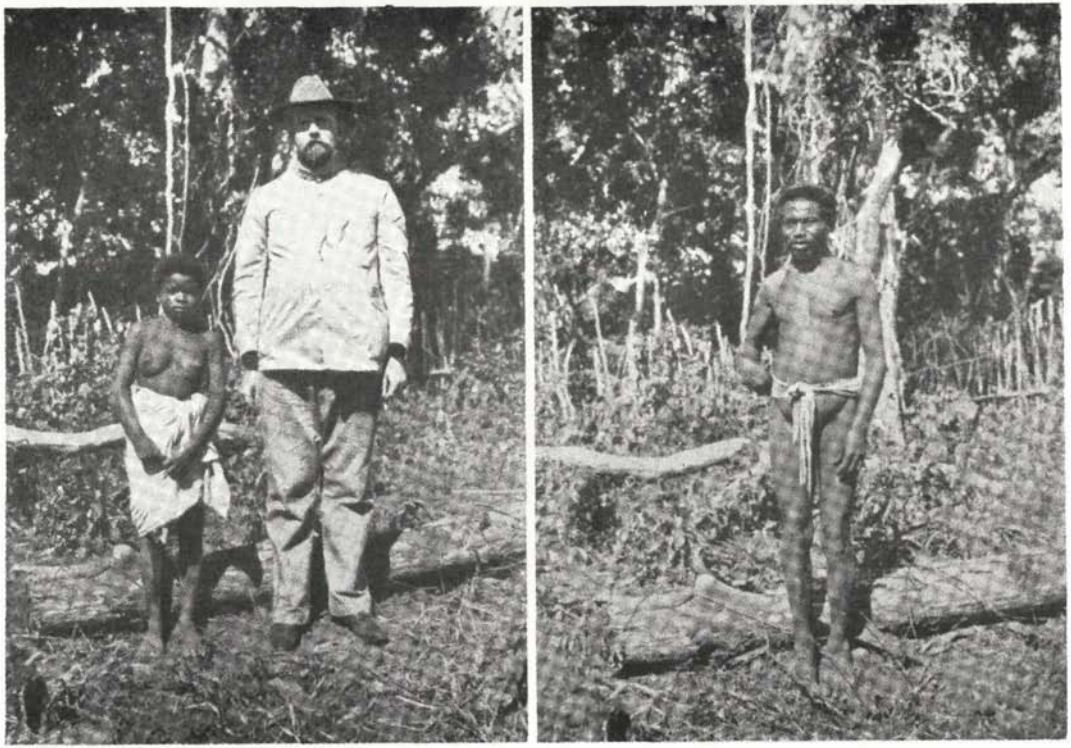

FIGURE 23—Southeast Asian Negritoes. Monte Mariveles Negritoes from Luzon in the Philippine Islands (Biasutti, 1959).

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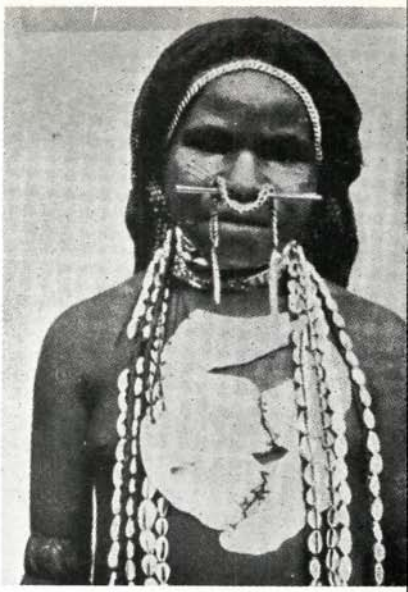

FIGURE 24—Melanesian Negroids. Ghende man (left) and Ghende woman from western New Guinea (Biasutti, 1959).

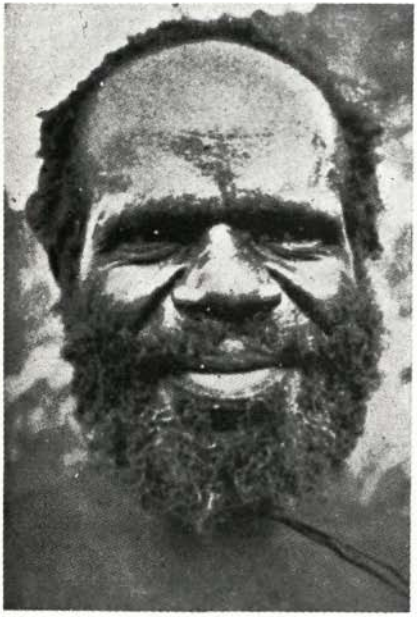

FIGURE 25—Southeast Asian Australoids. Ekari man (left) and Ekari woman from western New Guinea (Biasutti, 1959).

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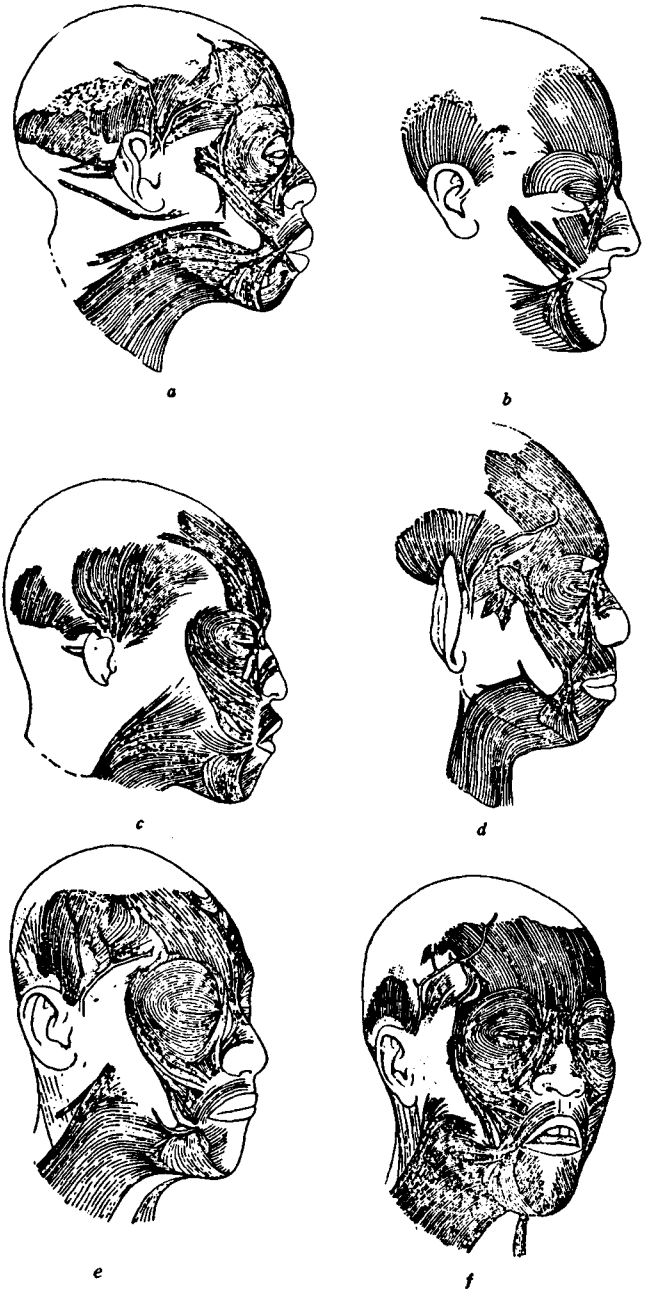

FIGURE 2—Racial differences in muscles of the face (Canella, 1943). a, African Negro; b, European White; c, Papuan; d, Australian Aborigine; e, Polynesian; f, Chinese.

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Book Reviews

THE BIRTH OF JAPAN

By Tingsen Shenfu Wei. China Press, Taiwan. Received for review 1976. Numerous figures and diagrams. Pp. 353.

Although recently published, this is after a long delay, and carries, as a result, a foreword by Professor R. Ruggles Gates. He says that to have solved the problem of the origin of Jimmu Tenno, the first Japanese Emperor, is a convincing achievement. Professor Tingsen Shenfu Wei shows that Jimmu Tenno was Hsü Fu of Chinese history, descended from a Chinese Prince of Hsü. In the time of Jimmu Tenno, who came with an expedition from China, the natives of Japan were a people of Neolithic (Jomon) culture and pottery of simple corded type. This type would appear to make its appearance between B.C. 3000 and B.C. 2500 approximately by radio-carbon dating.

The book covers the whole of the archaeology of Japan from the palaeolithic and mesolithic cultures onwards and right through to historic times, and has also a useful bibliography. This is a useful book, especially for Western scholars, for whom works on Japan are not readily accessible because of linguistic difficulties.

R. GAYRE OF GAYRE

CIVILIZED MAN'S EIGHT DEADLY SINS

By Konrad Lenz. Methuen, London, 1974. Pp. 80. Price £1.10 (hardback), 60p (paperback).

This book says what has needed to be said for a long time. Lenz, a Nobel prizewinner, summarises the position under eight heads. Over-population forces man to shut himself off in an "inhuman" way which elicits aggression. Devastation of our environment destroys the beauty which induces in man a reverential awe for a creation superior to himself. Man's race against himself robs man of his genuinely human activity of reflection. With this goes the warning of strong feelings and emotion caused by self-indulgence; and joy and sorrow ebb away into unutterable boredom. Genetic decay takes the form of infantilism, and this is seen in a certain type of hippie parasite. This also is associated with a break with tradition which comes about when the younger generation can no longer communicate with the older. With this goes the increased indoctrinability of mankind. With this goes the arming of mankind with nuclear weapons, which constitute a threat easier to avert than the seven other developments described.

In his chapter on genetic decay he has this to say of progress infantilism of our times: "Countless young people are hostile to modern society and to their parents. The fact that in spite of this attitude they still expect to be kept by this society and their parents shows their unreflecting infantilism" (p. 43).

This book should be read widely.

R. GAYRE OF GAYRE

GENETICS, EVOLUTION AND MAN

By W. F. Bodmer and L. L. Cavalli-Sforza. W. H. Freeman and Company Ltd. San Francisco, California, and Reading, England. Pp. 782, illustrated by many maps, figures and tables. Price $14.95 (£11.10).

Although this is described as an introductory book, it will be seen from its size that it is an exhaustive work. It covers the whole range of genetics population genetics, and evolution. It deals with modern man, and so it is relevant to anthropology.

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The work is compiled by Professor Bodmer, Chair of Genetics at Oxford, and Professor L. L. Cavalli-Sforza, Professor of Genetics at Standford School of Medicine.

The writers use much effort in trying to argue the case against the difference between I.Q.s of Blacks and Whites (pp. 676 ff) as propounded by Jensen and Shockley. But the fact remains that all through pre-history and history the Blacks have been as backward, their difference in physical structure is clear, and they remain backward everywhere today, although in the U.S.A. for instance, they have no linguistic or essential educational barriers. The Scots and Jews in the U.S.A. came in in depressed circumstances (a part of the former as slaves after the defeat of the Jacobite army in 1745) but within one or two generations were monied people. They leave one with the impression that they employ many pages pointing out exceptions or a special pleading. No doubt this book will please the age in which it is written but will not stand, in my opinion, the test of time.

We believe the work of Burt, Herrnstein, Jensen, Shockley, Garrett, Gates, etc., along with that of the anthropologists such as Elliott Smith, and Keith, will be proved right in the end.

It is, nevertheless, a good book so far as it teaches genetics, and only becomes uncertain or muffled in its sound where it touches the problems of our times.

R. GAYRE OF GAYRE

THE CULTURAL CONTEXT OF LEARNING AND THINKING: AN EXPLORATION IN EXPERIMENTAL ANTHROPOLOGY

By Michael Cole, John Gay, Joseph A. Glick, and Donald W. Sharp. Tavistock Publications, London, June 1974. Pp. 304, map, illustrations and figures. Price £1.50 (paperback).

This book deals with the Kpelle of Liberia. It starts with a prejudice in the dedication: "We believe that there is no undoing the harm that Americans as a group have brought to our hosts." I would have thought that any harm was done by the Liberians to the Kpelle. However, putting that aside, it spends no time on the genetic nature of man, boosts Boas, and ends by some criticism of Jensen.

It does not tackle the self-evident problem that there are mental differences between the Caucasoids and Negroids, which are equally supported if we consider their failure (although having English from birth) in school statistics when compared with Pakistanis (who had none), or that now the Amerindian is getting education the American Negro is inferior to them when manipulating the nuances of Western thought and techniques.

ADOPTION IN EASTERN OCEANIA

Edited by Vern Carroll. University of Hawaii Press, Honolulu, 1970. Association for Social Anthropology in Oceania Monograph Series. Pp. 422, 33 tables, 24 figures.

This volume discusses the procedure which in Oceania a child may be taken as one's own (adopted) although it is born of other parentage. The contributors include, besides the editor, John L. Fischev, Ronald G. Gallimore, Ruth Gallagher Goodenough, Ward H. Goodenough, Robert H. Heighton, Antony Hooper, Alan Howard, Cathie E. Jordan, Bernd Lambert, Robert I. Levy, Michael D. Lieber, Henry P. Lundsgaarde, Paul Ohino, Harold W. Scheffler, and Martin Gary Silverman.

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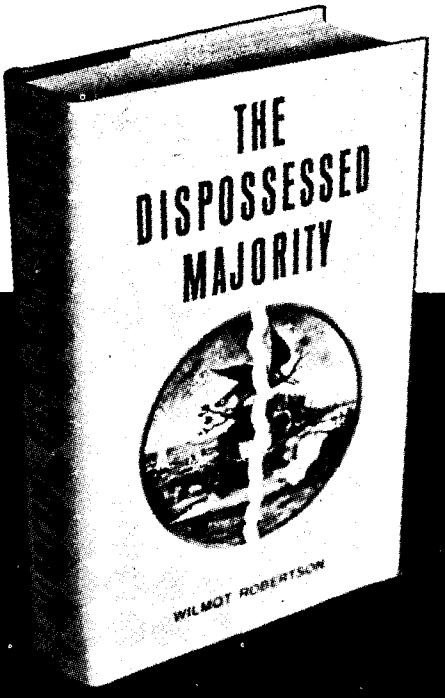

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